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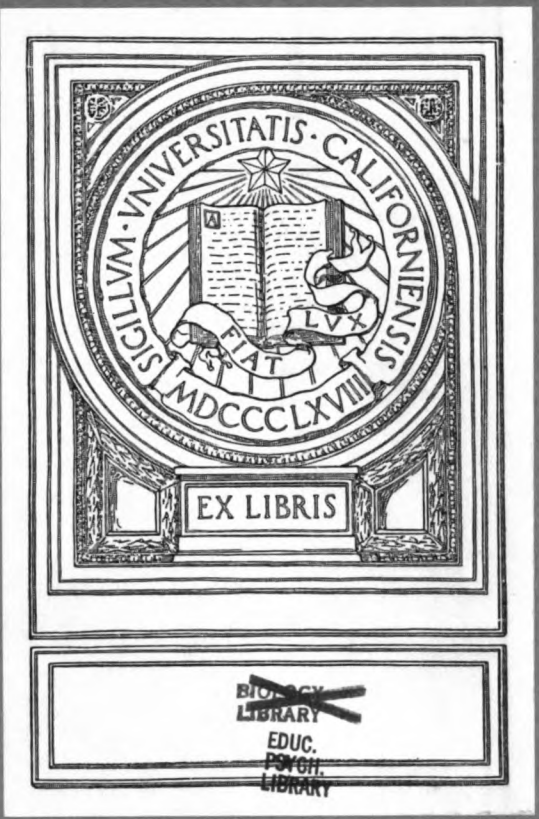

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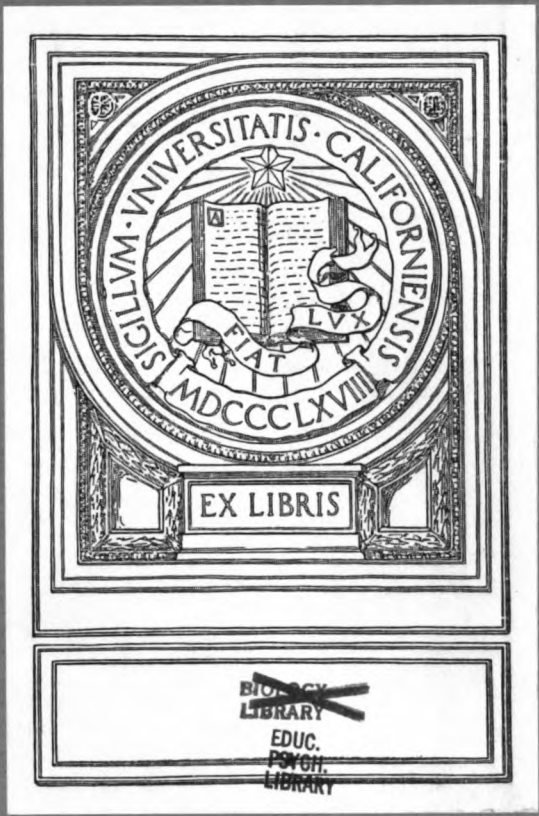

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OF

MENTAL SCIENCE

BY AUTHORITY OF
THE ROYAL MEDICO-PSYCHOLOGICAL ASSOCIATION

UNIV. OF
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" In adopting our title of the *Journal of Mental Science, published by authority of the Medico-Psychological Association*, we profess that we cultivate in our pages mental science of a particular kind, namely, such mental science as appertains to medical men who are engaged in the treatment of the insane. But it has been objected that the term mental science is inapplicable, and that the term mental physiology or mental pathology, or psychology, or psychiatry (a term much affected by our German brethren), would have been more correct and appropriate; and that, moreover, we do not deal in mental science, which is properly the sphere of the aspiring metaphysical intellect. If mental science is strictly synonymous with metaphysics, these objections are certainly valid; for although we do not eschew metaphysical discussion, the aim of this JOURNAL is certainly bent upon more attainable objects than the pursuit of those recondite inquiries which have occupied the most ambitious intellects from the time of Plato to the present, with so much labour and so little result. But while we admit that metaphysics may be called one department of mental science, we maintain that mental physiology and mental pathology are also mental science under a different aspect. While metaphysics may be called speculative mental science, mental physiology and pathology, with their vast range of inquiry into insanity, education, crime, and all things which tend to preserve mental health, or to produce mental disease, are not less questions of mental science in its practical, that is in its sociological point of view. If it were not unjust to high mathematics to compare it in any way with abstruse metaphysics, it would illustrate our meaning to say that our practical mental science would fairly bear the same relation to the mental science of the metaphysicians as applied mathematics bears to the pure science. In both instances the aim of the pure science is the attainment of abstract truth; its utility, however, frequently going no further than to serve as a gymnasium for the intellect. In both instances the mixed science aims at, and, to a certain extent, attains immediate practical results of the greatest utility to the welfare of mankind; we therefore maintain that our JOURNAL is not inaptly called the *Journal of Mental Science*, although the science may only attempt to deal with sociological and medical inquiries, relating either to the preservation of the health of the mind or to the amelioration or cure of its diseases; and although not soaring to the height of abstruse metaphysics, we only aim at such metaphysical knowledge as may be available to our purposes, as the mechanician uses the formularies of mathematics. This is our view of the kind of mental science which physicians engaged in the grave responsibility of caring for the mental health of their fellow-men may, in all modesty, pretend to cultivate; and while we cannot doubt that all additions to our certain knowledge in the speculative department of the science will be great gain, the necessities of duty and of danger must ever compel us to pursue that knowledge which is to be obtained in the practical departments of science with the earnestness of real workmen. The captain of a ship would be none the worse for being well acquainted with the higher branches of astronomical science, but it is the practical part of that science as it is applicable to navigation which he is compelled to study."—*Sir J. C. Bucknill, M.D., F.R.S.* (*Journ. Ment. Sci.*, vol. vii, 1861, p. 137).

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TRAINING INSTITUTIONS FOR NURSES
HONOURS ROLL
MEDALS, PRIZES, AND CERTIFICATES
LIST OF MEMBERS, ETC.

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List of Examination Areas and Area Examination Secretaries for the Mental Nursing Certificate.

SOUTH-EASTERN DIVISION: (1) Northern Area (North of Thames), W. J. T. KIMBER; (2) Southern Area (South of Thames), A. A. W. PETRIE.

SOUTH-WESTERN DIVISION: (1) Bristol Area, J. J. B. MARTIN; (2) Cardiff Area N. MOULSON; (3) Exeter Area, D. McKINLEY REID; (4) Oxford Area, J. F. W. LEECH; (5) Salisbury Area, C. W. S. DAVIES-JONES.

NORTHERN AND MIDLAND DIVISION: (1) Nottingham Area, J. BAIN; (2) Manchester Area, H. DOVE CORMAC; (3) Leeds Area, C. W. EWING; (4) York Area, T. M. DAVIE; (5) Newcastle Area, H. G. DRAKE-BROCKMAN; (6) Birmingham Area, C. W. FORSYTH.

SCOTTISH DIVISION: (One Area) A. M. DRYDEN.

IRISH DIVISION (One Area): KATHLEEN DILLON.

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Training Institutions for Nurses.

[For recognition as Training Schools, Mental Institutions and Institutions for Mental Defectives must be visited and reported on favourably by a referee or referees appointed by the Training Sub-Committee or Registrar.]

FOR TRAINING MENTAL NURSES.

(1) ENGLAND AND WALES.

(i) All County, City, and Borough Mental Hospitals, including Tooting Bec Hospital.

(ii) Criminal Asylum: Broadmoor.

(iii) Registered Hospitals: St. Andrew's (Northampton), Manchester Royal, Holloway Sanatorium, Bethlem Royal, The Retreat, Barnwood House, Bootham Park, Coton Hill, Wonford House, The Warneford, The Coppice, The Lawn (for Female Nurses only).

(iv) Special Hospitals: Netley (D Block); Royal Naval Hospital, Yarmouth; Maudsley Hospital; St. George's Retreat, Burgess Hill, Sussex (with the Hospital of St. John and St. Elizabeth, for Training in Medical and Surgical Nursing).

(v) Private Mental Hospitals: The Old Manor, Camberwell House, Laverstock House, Peckham House, Brislington House, Ticehurst House, Northumberland House, The Priory, Middleton Hall, Bailbrook House, Brooke House (for Female Nurses only).

(vi) Municipal Hospitals: Erdington House (Birmingham), Smithdown Road (Liverpool).

(2) SCOTLAND.

(i) Royal Asylums and Hospitals: Aberdeen, Crichton (Dumfries), Dundee, Edinburgh, Glasgow (Gartnavel), Montrose, James Murray's (Perth).

(ii) All City and District Asylums and Mental Hospitals.

(iii) Poor Law Asylum: Greenock.

(iv) Poor Law Hospital: Stobhill Hospital (Glasgow).

(v) Private Mental Hospital: New Saughton Hall (Polton).

(3) IRELAND.

(i) All County, City and District Asylums and Mental Hospitals.

(ii) Criminal Asylum: Dundrum.

(iii) Chartered Hospitals: St. Patrick's.

(iv) Private Mental Hospital: Farnham House (for Female Nurses only).

(4) CHANNEL ISLANDS.

Jersey Mental Hospital.

(5) FEDERATED MALAY STATES.

Central Mental Hospital, Tanjong Rambutan, Perak.

(6) INDIA.

Ranchi Mental Hospital for Europeans, Kankè, Province of Bihar and Orissa.

(7) SOUTHERN RHODESIA.

Ingutshihi.

FOR TRAINING THOSE NURSING MENTAL DEFECTIVES.

(1) ENGLAND.

(i) State Institutions: Rampton, Moss Side.

(ii) Certified Institutions: Botley's Park, Surrey; Brentry; Brunswick House, Mistley, Essex (for Male Nurses); Calderstones; Clerks Croft, Surrey; Cell Barnes Colony, Herts; Coldeast Colony, near Southampton; Coleshill Hall; Farmfield: Great Barr Park Colony; Harmston Hall Colony, near Lincoln; Hensol Castle, Pontyclun, Glam.; Hortham, Bristol; Leybourne Grange Colony; Monyhull Colony; Pewsey Colony, Wilts; Royal Albert Institution; Royal Earlswood; Royal Eastern Counties; Sandhill Park (with ancillary premises); Silverton House, Northumberland (for Female Nurses); South Ockenden Colony, Essex; South Side, Streatham; The Manor, Epsom; Middlesex Colony, Shenley; Western Counties Institution; Little Plumstead Hall Colony (with Heckingham ancillary premises), Norfolk; Stretton Hall, Leicester (for Female Nurses); Brockhall, Blackburn; Stoke Park Colony, Bristol; Cranage Hall, Holmes Chapel, Cheshire.

(iii) Approved Institutions: Darenth, Leavesden, Caterham, Fountain (for Female Nurses only).

(iv) Certified Houses: Normansfield.

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(2) SCOTLAND.

Baldovan; Gogarburn, Edinburgh; Royal Scottish National Institution; Stoneyetts.

FOR TRAINING FOR THE CERTIFICATE OF OCCUPATIONAL THERAPY.

ENGLAND.

North Riding Mental Hospital, York.

The Library and Secretary's Office.

11, CHANDOS STREET, LONDON, W. 1.

Telephone: Langham 1495.

Secretary and Assistant Librarian.—Miss M. HIGGINS.

Publications.

From MESSRS. ADLARD & SON, LTD.:

Year Book of the Association. Price 6*d*. Post free.
The Charter and Bye-Laws of the Association. Price 1/-. Post free.
Engraved Book-Plate of the Association. Price 1/-. Post free.
Portrait of Dr. Samuel Hitch. Price 1/-. Post free.
" The Clinical Study of Mental Disorders." Price 6/-. Post free.
" First Report on the Comparative Incidence of Cancer in Mental Hospital Patients and in the General Population of England." Price 9*d*. Post free.
" The State Registration of Mental Nurses." Parts I and II. Price 6*d*. each. Post free.
" An Account of a Tour of Dutch Mental Hospitals and Clinics." Price 1/-. Post free.
Enquiry (History) Form for use in Mental Hospitals.

From MESSRS. J. & A. CHURCHILL, LTD:

" The Journal of Mental Science." Published six times a year. Price 6/-. Postage extra.
General Index of the Journal, vol. lv to lxviii (inclusive). Price 2/6. Post free.
Binding Cases for the General Index, price 2/- (post free), are obtainable from Messrs. Adlard & Son, Ltd., who also undertake binding, including case, for 4/6.
Catalogue of the Association's Library. Price 2/-. Post free.

From MESSRS. BAILLIÈRE, TINDALL & COX:

" Handbook for Mental Nurses." 7th edn. 1923. Price 6/- net.
" Manual for Mental Deficiency Nurses." 1931. Price 6/- net.

From the REGISTRAR, St. Andrew's Hospital, Northampton:

" Regulations " } for the Examinations for the Certificates of Proficiency in Mental Nursing
" Rules " } and in Nursing Mental Defectives. Price 2*d*. each. Postage ½*d*.
" Syllabuses " }
" Syllabuses ", for the Examination for the Certificate of Proficiency in Occupational Therapy. Price 2*d*. each. Postage ½*d*.

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Honours Roll.

Year.	Gaskell Medal and Prize.	Bronze Medal and Prize.
1883	—	J. Wiglesworth.*
1884	—	S. R. Macphail.
1885	—	T. D. Greenlees.*
1886	—	—
1887	J. E. D. Mortimer.	J. Wiglesworth.
1888	—	—
1889	T. B. Hyslop.	—
1890	G. M. Robertson.	J. C. Mackenzie. W. J. Smythe.
1891	—	—
1892	Nathan Raw. G. R. Wilson.	G. M. Robertson.
1893	—	A. W. Campbell.
1894	—	C. Beadles.
1895	G. W. F. Macnaughton.	—
1896	W. R. Dawson.	J. Turner.
1897	C. Hubert Bond.	—
1898	—	J. R. Lord.
1899	—	F. G. Crookshank.
1900	Maurice Craig.	C. C. Easterbrook.
1901	W. H. B. Stoddart.	—
1902	G. H. Grills.	—
1903	—	—
1904	—	A. A. D. Townsend.
1905	—	—
1906	J. M. Rutherford.	C. J. Shaw.
1907	—	—
1908	—	S. C. Howard.
1909	H. Devine.	C. F. F. McDowall.
1910	—	H. Morton.
1911	J. G. P. Phillips.	G. H. Garnett.
1912	W. Boyd.	—
1913	W. R. Thomas.	D. McKinley Reid.
1914	—	J. C. Wootton.
1915	—	—
1916	—	—
1917	J. C. Woods.	—
1918	—	—
1919	J. Walker.	R. Eager.
1920	—	—
1921	—	—
1922	—	W. S. Dawson.
1923	J. T. H. Madill.	—
1924	Mary Barkas.	—
1925	W. S. Dawson.	—
1926	G. de M. Rudolf.	P. K. McCowan.
1927	Elizabeth Casson.	—
1928	F. R. Martin.	—
1929	Alexander Walk.	—
1930	G. W. T. H. Fleming.	Duncan Macmillan.
1931	—	J. H. Ewen.
1932	Muriel L. M. Northcote.	W. R. Ashby.
1933	Louis Minski. Desmond Curran.	D. N. Parfitt.
1934	William Blyth.	H. C. Beccle.
1935	—	Arthur Guirdham.
1936	S. W. Hardwick.	D. K. Bruce.

* Prize only.

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Medals and Prizes.

The Gaskell Gold Medal and Prize.

(Value not less than £30.)

This Medal and Prize was established, at the Annual Meeting held on August 9, 1886, in honour of Samuel Gaskell, Esq., F.R.C.S., at one time Medical Superintendent of the County Asylum, Lancaster, and afterwards for 17 years a Commissioner in Lunacy. Upon his death on March 16, 1886, his sister, Mrs. Holland, gave £1000 to the Association, and other members of the family contributed further sums amounting to £340. The interest of this sum is given annually as a prize to the candidate who most distinguishes himself in an Examination in the subjects hereunder mentioned.

The examination must be held in England according to the terms of the Trust.

(1) Candidates must produce evidence: (*a*) Of having attained the age of 23. (*b*) Of having been qualified Medical Officers in one or more mental hospitals or clinics in psychiatry in the United Kingdom or elsewhere in the British Empire for at least two years. (*c*) Of possessing the Certificate in Psychological Medicine of this Association or a degree or diploma in Psychological Medicine of one of the Universities or Examining Boards having the power to grant medical qualifications registrable in the British Isles or elsewhere in the British Empire.

(2) Candidates will be examined in: (*a*) Psychology. (*b*) Anatomy, Histology and Physiology of the Nervous System, reproductive and endocrine glands; Pathology of the above, especially in relation to mental disorders. (*c*) The diagnosis, prognosis, treatment and legal relations of mental disorders. (*d*) Clinical cases.

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The Bronze Medal and Prize.

(Value Ten Guineas.)

The Bronze Medal and Prize was established at the Annual Meeting held on August 2, 1882, and is awarded to any assistant medical officer of a mental hospital (public or private) for the best dissertation on a clinical or pathological subject relating to mental disorders.

The following is an abstract of the conditions:

(1) The dissertation to be written in English.

(2) Each dissertation to be distinguished by a motto or device, and accompanied by a sealed envelope containing the name and residence of the author, and having on the outside a motto or device corresponding with that on the dissertation.

(3) The dissertation shall not exceed 10,000 words in length.

(4) The dissertation and every accompanying drawing and preparation will become the property of the Association, to be published in the Journal at the discretion of the Editors.

(5) The dissertations not approved, with their accompanying drawings and preparations, will, upon application within one year, be returned, together with the sealed envelopes unopened; if not claimed within one year they will become the property of the Association.

(6) The dissertations for the year must be delivered to the Registrar before April 30.

(7) No prize will be awarded if none of the dissertations is of sufficient merit.

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Divisional Prizes for Assistant Medical Officers.

(Two Prizes of £10 and £5 respectively.)

The proposal to award Divisional Prizes under the conditions set out below emanated from the Irish Division and was adopted at the Annual Meeting held on July 13, 1911.

(1) Two prizes of £10 and £5 respectively will be awarded annually by the Association (provided sufficient merit be shown) for the best paper read during the preceding calendar year at a Divisional Meeting by an Assistant Medical Officer or Assistant Physician in a Psychiatric or Neurological Institution or Service. (*See also Regulation* No. 7.)

(2) Competitors must be members of the Association.

(3) Only one paper by a competitor can be entered during any competitive year. The same applies to a paper entered by more than one author.*

(4) Only papers certified by the Secretary of the Division (in the absence of the Secretary, the Chairman of the Meeting) to have been read at a meeting of the Division and to be eligible for this competition can be entered. Divisional Secretaries are to withhold their certificates in cases where there has occurred material alterations or additions.

(5) Papers certified as eligible for the competition shall be forwarded to the Registrar not later than April 30 of the following year. They shall be submitted to the Examiners for the Certificate in Psychological Medicine for report and then adjudicated on by the President. Publication shall not invalidate a paper for adjudication.†

(6) The results shall be announced, and the prizes awarded, if any, by the President at the Annual Meeting in the year following the competitive year.

(7) Papers by more than one author can be entered and any prize awarded be divided among the authors.

(8) If in any competitive year one or two papers only are submitted for competition the Council may withhold either or both prizes; or it may, after considering the report of the Adjudicator, and if in its opinion the paper or papers are of outstanding merit, award a first or second or both prizes, at its discretion.

The Certificate in Psychological Medicine.

This Certificate was founded at the Annual Meeting held on August 4, 1885, and the first examinations were held in 1886.

Candidates must be at least twenty-one years of age, and must produce a certificate of having at least three months' clinical experience of mental disorders, or of having attended a course of lectures on insanity and the practice of a mental hospital (where there is clinical teaching) for a like period, or they shall give such proof of experience in mental disorders as shall, in the opinion of the President, be sufficient.

The examination is held yearly, and is in three parts: (1) Written. (2) Oral. (3) Practical.

Candidates must be registered under the Medical Act (1858) before the certificate is bestowed.

The fee for the examination is £3 3s., and intending candidates should communicate with the Registrar, who will supply date and place of examination.

Candidates failing in the examination may present themselves at the next or subsequent examinations on payment of a fee of £1 1s.

* This means that the joint authors (though separately eligible) cannot enter another paper in the competitive year.

† The original MS., certified in accordance with Regulation No. 4, is to be submitted. Only under exceptional circumstances can a certified reprint of a published paper be accepted, but a reprint, where possible, should accompany the certified original MS.

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The Insignia of the Nursing Certificate.

The Certificate of Proficiency in Mental Nursing was founded at the Annual Meeting held on July 24, 1890.

The Nursing Medal.

The Nursing Badge.

The Nursing Badge was founded at the Annual Meeting held on July 11, 1928.

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Honorary Certificate in Mental Nursing.

1928. H.R.H. The Princess Royal.

The Campbell Clark Gold Medal and Prize.

This Prize was instituted in 1933 in memory of the late Dr. Campbell Clark, and is awarded in May and November of each year to the candidate, whether male or female, who obtains the highest number of marks at the Examination for the Certificate of Proficiency in Mental Nursing. It is provided that only candidates who have completed the whole of their training in one institution are eligible. The value of the prize is Three Guineas, together with the Medal.

Winners of the Campbell Clark Medal.

1933, May. Arthur Barnett Hindson, Cambridge Mental Hospital.
,, Nov. Florence Barber, Maudsley Hospital.
1934, May. Cissie Morris, Bexley Mental Hospital.
,, Nov. John Humphries, Belfast Mental Hospital.
1935, May. Thelma Mary Preece, Powick Mental Hospital.
,, Nov. Ronald Nears, Claybury Mental Hospital.
1936, May. Winifred Amelia Hill, Erdington House.

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List of Chairmen.

1841. Dr. Blake, Nottingham General Asylum.
1842. Dr. de Vitré, Lancaster Asylum.
1843. Dr. Conolly, Hanwell Asylum.
1844. Dr. Thurnam, The Retreat.
1847. Dr. Wintle, Warneford House.
1848. William Ley, M.R.C.S., Littlemore Asylum.
1851. Dr. Conolly, Hanwell Asylum.
1852. Dr. Wintle, Warneford House.

List of Presidents.

1854. A. J. Sutherland, M.D., St. Luke's Hospital, London.
1855. J. Thurnam, M.D., Wilts County Asylum.
1856. J. Hitchman, M.D., Derby County Asylum.
1857. Forbes Winslow, M.D., Sussex House, Hammersmith.
1858. John Conolly, M.D., Hanwell Asylum.
1859. Sir Charles Hastings, D.C.L., Worcester.
1860. J. C. Bucknill, M.D., Devon County Asylum.
1861. Joseph Lalor, M.D., Richmond Asylum, Dublin.
1862. John Kirkman, M.D., Suffolk County Asylum.
1863. David Skae, M.D., Royal Asylum, Edinburgh.
1864. Henry Munro, M.D., Brook House.
1865. Wm. Wood, M.D., Kensington House.
1866. W. A. F. Browne, M.D., Commissioner in Lunacy for Scotland.
1867. C. A. Lockhart Robertson, M.D., Haywards Heath Asylum.
1868. W. H. O. Sankey, M.D., Sandywell Park, Cheltenham.
1869. T. Laycock, M.D., Edinburgh.
1870. Robert Boyd, M.D., Wells Asylum.
1871. Henry Maudsley, M.D., The Lawn, Hanwell.
1872. Sir James Coxe, M.D., Commissioner in Lunacy for Scotland.
1873. Harrington Tuke, M.D., Chiswick House.
1874. T. L. Rogers, M.D., Rainhill Asylum.
1875. J. F. Duncan, M.D., Dublin.
1876. W. H. Parsey, M.D., Warwick County Asylum.
1877. G. Fielding Blandford, M.D., London.
1878. Sir J. Crichton-Browne, M.D., Lord Chancellor's Visitor in Lunacy.
1879. J. A. Lush, M.D., Fisherton House.
1880. G. W. Mould, M.R.C.S., Royal Asylum, Cheadle.
1881. D. Hack Tuke, M.D., London.
1882. Sir W. T. Gairdner, M.D., Glasgow.
1883. W. Orange, M.D., State Criminal Lunatic Asylum, Broadmoor.
1884. Henry Rayner, M.D., Hanwell Asylum.
1885. J. A. Eames, M.D., District Asylum, Cork.
1886. Sir Geo. H. Savage, M.D., Bethlem Royal Hospital.
1887. Sir Fred. Needham, M.D., Barnwood House.
1888. Sir T. S. Clouston, M.D., Royal Asylum, Edinburgh.
1889. H. Hayes Newington, F.R.C.P., Ticehurst House.
1890. David Yellowlees, M.D., Royal Asylum, Glasgow.
1891. E. B. Whitcombe, M.R.C.S., City Asylum, Birmingham.
1892. Robert Baker, M.D., The Retreat.
1893. J. Murray Lindsay, M.D., Derby County Asylum.
1894. Conolly Norman, F.R.C.P.I., Richmond Asylum, Dublin.
1895. David Nicolson, *C.B.*, M.D., State Criminal Lunatic Asylum, Broadmoor.
1896. Willi am Julius Mickle, M.D., Grove Hall.
1897. Thomas W. McDowall, M.D., Northumberland Asylum.
1898. A. R. Urquhart, M.D., James Murray's Royal Asylum, Perth.
1899. J. B. Spence, *O.B.E.*, M.D., Burntwood Asylum, Lichfield.
1900. Fletcher Beach, M.B., London.
1901. Oscar T. Woods, M.D., District Asylum, Cork.
1902. J. Wiglesworth, M.D., Rainhill Asylum.

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1903. Ernest W. White, *C.B.E.*, M.B., City of London Asylum, Stone.
1904. R. Percy Smith, M.D., London.
1905. T. Outterson Wood, M.D., London.
1906. Sir Robert Armstrong-Jones, *C.B.E.*, M.D., Claybury Asylum.
1907. P. W. MacDonald, M.D., Dorchester County Asylum.
1908. Chas. A. Mercier, M.D., Flower House.
1909. W. Bevan-Lewis, M.Sc., West Riding Asylum, Wakefield.
1910. Sir John Macpherson, M.D., Commissioner in Lunacy for Scotland.
1911. Wm. R. Dawson, *O.B.E.*, M.D., Inspector of Lunatic Asylums, Dublin.
1912. J. Greig Soutar, M.B., Barnwood House.
1913. James Chambers, M.D., The Priory.
1914–18. David G. Thomson, *C.B.E.*, M.D., Norfolk County Asylum.
1918. John Keay, *C.B.E.*, M.D., Bangour Village.
1919. Bedford Pierce, M.D., The Retreat.
1920. William F. Menzies, M.D., Cheddleton Mental Hospital.
1921. Sir Hubert Bond, *K.B.E.*, M.D., Commissioner, Board of Control.
1922. G. M. Robertson, M.D., Royal Mental Hospital, Morningside, Edinburgh.
1923. Edwin Goodall, *C.B.E.*, M.D., Cardiff City Mental Hospital.
1924. Michael J. Nolan, L.R.C.P.&S.Irel., Downpatrick Asylum.
1925. Sir Frederick W. Mott, *K.B.E.*, M.D., London.
1926. John R. Lord, *C.B.E.*, M.D., Horton Mental Hospital, Epsom.
1927. Hamilton Marr, *C.B.*, M.D., Commissioner, General Board of Control for Scotland.
1928. J. Shaw Bolton, D.Sc., M.D., West Riding Mental Hospital, Wakefield.
1929. Nathan Raw, *C.M.G.*, M.D., F.R.C.S.E., F.R.S.E., Lord Chancellor's Visitor in Lunacy.
1930. T. Saxty Good, *O.B.E.*, M.A., M.R.C.S., L.R.C.P., Oxford County and City Mental Hospital, Littlemore.
1931. R. R. Leeper, L.R.C.P.I., F.R.C.S.I., St. Patrick's Hospital, Dublin.
1932. R. B. Campbell, M.D., F.R.C.P.E., Stirling District Mental Hospital, Larbert.
1933. Frank Douglas Turner, M.B., M.R.C.S., L.R.C.P., Royal Eastern Counties Institution, Colchester.
1934. Daniel F. Rambaut, M.A., M.D., St. Andrew's Hospital, Northampton.
1935. Reginald Worth, *O.B.E.*, M.B., Springfield Mental Hospital.
1936. M. A. Collins, *O.B.E.*, M.D., B.S., M.R.C.S., L.R.C.P., Canterbury.

List of Maudsley Lecturers.

1920. Sir J. Crichton-Browne, LL.D., D.Sc., M.D., F.R.S.
1921. Sir F. W. Mott, *K.B.E.*, LL.D., M.D., F.R.C.P., F.R.S.
1922. Sir M. Craig, *C.B.E.*, M.A., M.D., F.R.C.P.
1923. Charles Kirk Clarke, LL.D., M.D.
1924. J. Carswell, F.R.F.P.S.
1925. Joseph Shaw Bolton, D.Sc., M.D., F.R.C.P.
1926. George M. Robertson, M.D., F.R.C.P.E., F.R.C.S.E.
1927. Edwin Goodall, *C.B.E.*, M.D., F.R.C.P.
1928. Sir John Macpherson, *C.B.*, M.D., F.R.C.P.E.
1929. Charles E. Spearman, Ph.D., F.R.S.
1930. The Hon. Mr. Justice McCardie.
1931. Sir Hubert Bond, *K.B.E.*, LL.B., D.Sc., M.D., F.R.C.P.
1932. Sir Edward Farquhar Buzzard, Bart., *K.C.V.O.*, M.A., M.D., F.R.C.P.
1933. Adolf Meyer, M.D.
1934. The Right Hon. Lord Macmillan, M.A., LL.D.
1935. Lewis C. Bruce, *M.C.*, M.D., F.R.C.P.E., M.P.C.
1936. Sir Walter Langdon Brown, M.A., M.D., F.R.C.P.

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Membership of the Association.

South-Eastern Division:		
Honorary	15	343
Ordinary	328	
South-Western Division:		
Honorary	4	134
Ordinary	130	
Northern and Midland Division:		
Honorary	...	191
Ordinary	191	
Scottish Division:		
Honorary	1	102
Ordinary	101	
Irish Division:		
Honorary	1	73
Ordinary	72	
India:		
Ordinary	13	13
Other Countries:		
Honorary	16	113
Corresponding	51	
Ordinary	46	
Total Membership:		
Honorary	37	969
Corresponding	51	
Ordinary	881	

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HONORARY MEMBERS.

1932. Armstrong-Jones, Sir Robert, *C.B.E.*, D.Sc., M.D., B.S., F.R.C.P., F.R.C.S., F.S.A., *D.L., J.P.*, 27, Phillimore Gardens, W. 8 (and Plas Dinas, Carnarvon, North Wales). (*Ord. Mem. since* 1882; *Gen. Sec.*, 1897–1906; PRESIDENT, 1906–7.)

1928. Bleuler, Eugen, Prof. Dr., Zollikerstrasse 98, Zollikon bei Zürich, Switzerland.

1900. Blumer, G. Alder, M.D., L.R.C.P.Edin., Superintendent-Emeritus, Butler Hospital; 196, Blackstone Boulevard, Providence, R.I., U.S.A. (*Ord. Mem.*, 1890.)

1928. Bond, Sir Hubert, *K.B.E.*, D.Sc., M.D.Edin., LL.B.Lond., F.R.C.P., M.P.C., Senior Commissioner, Board of Control, Metropole Buildings, Northumberland Avenue, W.C. 2. Emeritus Lect. on Psychiat., Middlx. Hosp.) (*Ord. Mem.*, 1892; *General Secretary*, 1906–12; PRESIDENT, 1921–22.)

1900. Bresler, Johannes, Sanitätsrat Dr., Director of the Provincial Mental Hospital, Kreuzburg, Oberschlesien, Germany; Editor, *Psychiatrisch-neurologische Wochenschrift.* (*Corr. Mem.*, 1896.)

1932. Briggs, L. Vernon, M.D., 64, Beacon Street, Boston, Mass., U.S.A. (*Corr. Mem. since* 1923.)

1934. Brock, Sir L. G., *C.B.*, Chairman of the Board of Control, Metropole Buildings, Northumberland Avenue, W.C. 2.

1925. Chamberlain, Rt. Hon. Arthur Neville, *M.P.*, 37, Eaton Square, London, S.W. 1.

1931. Chambers, James, M.A., M.D., "Estia", Highdown Road, Roehampton, S.W. 15. (*Ord. Mem. since* 1888; *Asst. Editor*, 1900–05, and *Co-Editor of Journal*, 1905–14; PRESIDENT, 1913–14; *Treasurer*, 1917–31.)

1909. Collins, Sir Wm. Job, *K.C.V.O.*, B.Sc., M.D., M.S., F.R.C.S., 1, Albert Terrace, Regent's Park, London, N.W. 1.

1876. Crichton-Browne, Sir J., LL.D., D.Sc., M.D.Edin., F.R.S., 45, Hans Place, London, S.W. 1. (*Ord. Mem.*, 1863; PRESIDENT, 1878.)

1930. Cushing, Harvey, *C.B.*, A.M., LL.D., Sc.D., Litt.D., M.D., M.Ch., F.R.C.S., Professor of Surgery, The Yale Medical School, New Haven, Connecticut, U.S.A.

1936. Darwin, Ruth, Senior Commissioner, Board of Control, Metropole Buildings, Northumberland Avenue, W.C. 2.

1924. Dawson, Lt.-Col. W. R., *O.B.E.*, B.A., M.D., B.Ch.Dubl., F.R.C.P.Irel., D.P.H., M.P.C., 18, Brock Street, Bath. (*Ord. Mem.*, 1894; PRESIDENT, 1911–12; *Co-Editor of Journal*, 1920–21.)

1936. Doll, Edgar A., Ph.D., Director of Research at the Vineland Training School, Vineland, New Jersey; President of the American Association on Mental Deficiency.

1923. Ellis, Henry Havelock, L.S.A., 24, Holmdene Avenue, Herne Hill, S.E. 24.

1936. Freud, Sigmund, Prof. Dr., Wien 19, Strassergasse 47, Austria.

1922. l'Hermitte, Dr. Jacques Jean, Médecin de l'Hospice Paul Brousse, Paris; 9, rue Marbeuf, Paris (VIII^{e}), France.

1930. Janet, Pierre, M.D., Member of the Institute of France; Professor of Psychology, College of France; 54, Rue de Varenne, Paris (VII^{e}).

1930. von Jauregg, Hofrat Prof. Julius R. Wagner, M.D., late Professor of Psychiatry, University of Vienna; 1, Landesgerichtstr. 18, Vienna.

1935. Keay, John, *C.B.E.*, M.D., C.M., F.R.C.P., "Champions", Beaminster, Dorset. (*Ord. Mem.* 1886; PRESIDENT, 1918; *Chairman, Educational Committee*, 1920–27.)

1924. McDougall, William, LL.D., M.B., F.R.S., Professor of Psychology in Duke University, Durham, North Carolina, U.S.A.

1928. Macmillan, Rt. Hon. Lord, G.C.V.O., M.A., LL.D., Lord of Appeal in Ordinary; 44, Millbank, S.W. 1.

1910. Macpherson, Sir John, *C.B.*, M.D., C.M., F.R.C.P.Edin., "Hillside", Cleeve, nr. Bristol. (*Ord. Mem.*, 1886; PRESIDENT, 1910–11.)

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1921. Maudsley, Sir Henry Carr, *K.C.M.G.*, *C.B.E.*, M.D., B.S., F.R.C.P., Consulting Physician, Melbourne Hospital, Victoria; 8, Collins Street, Melbourne.
1926. Meyer, Adolf, M.D., Psychiatrist in Chief, Phipps Psychiatric Clinic, Johns Hopkins Hospital, Baltimore, Md., U.S.A.
1931. Nolan, Michael James, L.R.C.P.I., L.R.C.S.I., *J.P.*, Consulting Visitor-in-Lunacy to the Lord Chief Justice of Northern Ireland, and to the Chief Justice, Irish Free State; Slieve Dhu, Bryansford Road, Newcastle, Belfast, Ireland. (*Ord. Mem. since* 1888; PRESIDENT, 1924–5.)

1922. Pactet, Dr. François Florentin, Médecin Honoraire des Asiles de la Seine; Pavillon Colbert, Châtenay, Seine, France.

1927. Rolleston, Sir Humphry Davy, *Bt.*, *G.C.V.O.*, *K.C.B.*, D.C.L., LL.D., D.Sc., M.D., F.R.C.P., Physician Extraordinary to H.M. the King; "Martins", Scotlands Lane, Haslemere, Surrey.
1923. Rose, Lt.-Col. Sir Arthur, Bt., *D.S.O.*, Chairman, General Board of Control for Scotland, 25, Palmerston Place, Edinburgh.

1929. Sherrington, Sir Charles Scott, *O.M.*, *G.B.E.*, M.A., LL.D., D.Sc., M.D., F.R.C.P., F.R.C.S., F.R.S., Waynflete Professor of Physiology, Oxford; 9, Chadlington Road, Oxford.
1927. Smith, Robert Percy, M.D., B.S.Lond., F.R.C.P., 42, Albion Street, London, W. 2. (*Ord. Mem.*, 1885; *General Secretary*, 1896–97; *Chairman, Educational Committee*, 1899–1903; PRESIDENT, 1904–05.)

1901. Toulouse, Dr. Edouard, Médecin des Asiles de la Seine; 1, Rue Cabanis, Paris (XIV^{e}), France.

1934. Whitwell, James Richard, M.B.Edin., 95, Ormonde Court, S.W. 15. (*Ord. Mem. since* 1889; *Acting Hon. Librarian and Chairman, Library Committee, since* 1927; *Hon. Librarian since* 1928.)
1923. Willis, Sir Frederick James, *K.B.E.*, *C.B.*, Church House, Bramley, Guildford.
1926. Winkler, C., M.D., 35, Heerenstraat, Utrecht, Holland. (*Corr. Mem.*, 1924.)
1936. Wood, Rt. Hon. Sir Kingsley, M.P., Minister of Health, Whitehall, S.W. 1.

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CORRESPONDING MEMBERS.

1928. Bliss, Malcolm Andrews, M.D., Consultant in Neuro-Psychiatry, St. Luke's Hospital; 301, Himboldt Buildings, St. Louis, Minn., U.S.A.
1911. Boedeker, Geheimer Sanitätsrat, Prof. Justus Karl Edmund, Director, Zehlenhof Asylum; 18, Kolnrigs-Allee, Berlin-Grünewald.
1929. Bouman, K. Herman, M.D., Professor of Psychiatry and Director of the University Psychiatric Clinic, Jan Luykenstraat 24, Amsterdam.
1929. Buscaino, V. M., M.D., Professor of Psychiatry, University of Catania, Sicily.
1897. Buschan, Sanitätsrat Dr. med. et phil. G., Friedrich Carlstrasse 7, Stettin, Germany.

1930. Campbell, Charles Macfie, M.D., Professor of Psychiatry, Harvard Medical School; Director, Boston Psychopathic Hospital; 74, Fenwood Road, Boston, Massachusetts, U.S.A.
1927. Charpentier, René, M.D., Secrétaire Général de la Société Médico-Psychologique de Paris; 119, rue Perronet, Neuilly s/Seine (Seine), France.
1936. Cheney, Clarence O., Medical Director of the Westchester Division, New York Hospital, 121, Westchester Avenue, White Plains, New York, U.S.A.
1935. Combemale, Pierre, Professor of Psychiatry in the University of Lille, Médecin-Chef à l'Asile de Bailleul, Nord, France.
1904. Coroleŭ, Wilfrid, Medico forense del distrito de la Barceloneta, Aribau, 31, Barcelona, Spain.
1896. Cowan, F. M., M.D., 109, Perponcherstraat, The Hague, Holland.

1929. Ebaugh, Franklin G., A.B., M.D., Professor of Psychiatry, University of Colorado; Director, Colorado Psychiatric Hospital; 4200, East Ninth Avenue, Denver, Colorado, U.S.A.
1930. Evensen, Dr. Hans, Medical Director, Gaustad State Hospital, Vinderen, Norway; Commissioner in Legal Medicine for Norway; President, Norwegian Psychiatric Society.

1911. Falkenberg, Sanitätsrat, Dr. Wilhelm, Berlin-Friedenau, Ringstr. 5.
1907. Ferrari, Giulio Cesare, M.D., Director, Manicomio Provinciale, Imola, Bologna, Italy.
1930. Flournoy, Dr. Henri, 6, Rue de Monnetier, Geneva, Switzerland.
1911. Friedländer, Prof. Dr. Adolf Albrecht, Haus Sonnblick, Littenweiler, bei Freiburg i/Baden, Germany.

1931. Génil-Perrin, Dr., Chief Physician, Seine Department Mental Hospitals; General Secretary, French League for Mental Hygiene; 99, Avenue La Bourdonnais, Paris (VII^{e}).
1901. Gommès, Dr. Marcel, 5, Rue Parrot, Paris (XII^{e}).

1930. Haskovec, Prof. Dr. Ladislav, Professor of Nervous Diseases in the Charles University, Prague, Czecho-Slovakia.
1931. Hincks, Clarence Meredith, A.B., M.D., General Director of the National Committee for Mental Hygiene, 450, Seventh Avenue, New York, U.S.A.

1928. Kappers, C. U. Ariëns, M.D., Director, Netherlands Central Institution for Brain Research, Amsterdam.
1936. Klaesi, Prof. Jakob, Director, University Psychiatric Clinic, Waldau, Berne, Switzerland.
1934. Kraus, G., Provinciaal Ziekenhuis, Santpoort, by Haarlem, Holland.

1936. Maier, Prof. H. W., Director, University Psychiatric Clinic, Burghölzli, Zurich, Switzerland.
1932. May, James V., M.D., Medical Superintendent, Boston State Hospital, Dorchester Center Station, Boston, Mass., U.S.A.
1935. Mayer-Gross, W., Professor of Psychiatry in the University of Heidelberg; Rockefeller Research Fellow at the Maudsley Hospital.

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1930. Miyake, Dr. Koichi, Professor of Psychiatry, Tokio Imperial University; Director, Tokio Municipal Matsuzawa Psychopathic Hospital, Japan.
1931. Moreno, Dr. Samuel Ramirez, Director, Manicomio General, Mexico City.
1922. Morowoka, Dr. T., Owada, 102, Shibuya, Tokyo, Japan.

1929. Pameijer, J. H., M.D., Inspector of State Mental Hospitals, Bildersstraat 22, The Hague.
1928. Pighini, Giacomo, M.D., Professor of Psychiatry and Director, Laboratori Scientifici, Instituto Psichiatrico di S. Lazzaro, S. Maurizio, Reggio Emilia, Via De Amicis 10, Italy.
1909. Pilcz, Prof. Dr. Alexander, VIII/2 Alserstrasse 43, Vienna, Austria.
1929. Pratt, G. K., M.D., Assistant Medical Director, National Committee for Mental Hygiene; 370, Seventh Avenue, New York, U.S.A.

1931. Riedel, Dr. Gustavo, Director, Hospital-Psychopathic Colony of Engenbo de Dentro, Rio de Janeiro, Brazil; Hon. President, Brazilian League for Mental Hygiene; Vice-President, Brazilian Society of Psychiatry.
1931. Roemer, Dr. med. Hans, Director, Institute for Mental Diseases, Illenau, Baden, Germany; Secretary, German Association for Mental Hygiene; Secretary, Health Welfare Committee, Baden.
1931. Ruggles, Dr. Arthur H., Medical Superintendent, Butler Hospital, Providence, R.I., U.S.A.; Lecturer in Psychiatry, Yale University, New Haven; Chairman, Executive Committee of the National Committee for Mental Hygiene.

1922. Sano, Dr., Directeur de la Colonie de Gheel, Belgium.
1929. Scheer, Prof. W. M. van der, M.D., Psychiatrisch-Neurologische Kliniek, Groningen, Holland.
1934. Simon, Hermann, Direktor der Provinzialheilanstalt, Gütersloh, Westphalia, Germany.
1930. Strecker, Edward A., M.D., Professor of Nervous and Mental Diseases, Jefferson Medical College; 111, North Forty-ninth Street, Philadelphia, U.S.A.
1929. Stuurman, F. J., M.D., Secretary, Dutch Association for Psychiatry and Neurology; Oegstgeest, near Leyden, Holland.
1935. Sveinsson, Thordur, Medical Superintendent, Gamle Kleppur Asylum, Reykjavik, Iceland.

1927. Targowla, René, M.D., *Chev. Leg. Hon.*, 169, Rue de L'Université, Paris (VII^{e}).
1929. Thom, Douglas A., M.D., Professor of Mental Hygiene, Tuft Medical School; 520, Commonwealth Avenue, Boston, Mass., U.S.A.
1935. Tómasson, Helgi, Medical Superintendent, Nýja Spitalanum á Kleppi, Reykjavik, Iceland.

1930. Weygandt, Dr. phil. et med. Wilhelm, Professor of Psychiatry and Director, Psychiatric Clinic, University of Hamburg; Friedrichsbergerstr 60, Hamburg.
1929. Wiersma, E. D., late Professor of Psychiatry and Director of the University Psychiatric Clinic, Groningen, Holland.
1930. Williams, Frankwood E., D.Sc., M.D.; Editor, *Mental Hygiene and Mental Hygiène Bulletin;* 44, West 12th Street, New York City.
1928. Wimmer, August, M.D., Professor of Psychiatry, University of Copenhagen, Denmark.

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ORDINARY MEMBERS OF THE ASSOCIATION.

Alphabetical List of Ordinary Members of the Association on January 1, 1937, with the year in which they joined.

1928. Abd-el-Hakeem, Mohammed, M.B., B.Ch.Cairo, D.P.M., Assistant Medical Officer, Mental Hospital, Abbassia, Cairo, Egypt.

1910. Adam, George Henry, M.R.C.S., L.R.C.P., Resident Physician, Malling Place, West Malling, Kent.

1931. Adamson, James Weeden Woodhams, M.D.Durh., M.R.C.S., L.R.C.P., L.S.A., Commissioner of the Board of Control (England and Wales), Metropole Buildings, Northumberland Avenue, W.C. 2.

1919. Adey, J. K., M.B., C.M.Melb., Receiving House, Royal Park, Melbourne, Victoria, Australia.

1923. Ainsworth, Cyrus Gerald, M.A., LL.B., M.B., B.Ch.Camb., M.R.C.S., L.R.C.P., Medical Superintendent, Graylingwell Mental Hospital, Chichester.

1935. Aird, Patrick Joseph, M.B., B.Ch., B.A.O.N.U.I., D.P.M., D.P.H., Assistant Medical Officer, Grangegorman Mental Hospital, Dublin.

1931. Allan, Samuel Miller, M.D., Ch.B.Glasg., D.P.M., Assistant Medical Officer, Lancashire County Mental Hospital, Whittingham, Preston.

1932. Allen, James Stewart, M.B., B.Ch.Belf., B.Sc.Belf., D.P.M., Assistant Medical Officer, West Park Hospital, Epsom, Surrey.

1926. Albiston, Norman Arthur, M.B., B.S.Syd., D.P.M., 61, Collins Street, Melbourne, Australia.

1924. Alexander, Douglas Reid, *M.C.*, M.R.C.S., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, The Manor, Epsom, Surrey.

1922. Alexander, Marion Cameron, M.B., B.Ch., B.A.O.Belf., Dipl. Psych. Edin., Assistant Medical Officer, Storthes Hall Mental Hospital, Kirkburton, near Huddersfield.

1899. Allman, Dorah Elizabeth, M.B., B.Ch.R.U.I., Karavanagh, 52, Sandymount Avenue, Dublin.

1935. Anderson, Edward W., M.D., M.R.C.P., D.P.M., Medical Director, Cassel Hospital, Swaylands, Penshurst, Kent.

1929. Anderson, John Colquhoun, B.Sc., M.B., Ch.B.Glasg., Dipl. Psych., 68, Highburgh Road, Glasgow, W. 2.

1898. Anderson, John Sewell, M.R.C.S., L.R.C.P., Medical Superintendent, City Mental Hospital, Willerby, Hull.

1918. Anderson, William Kirkpatrick, M.D., Ch.B., F.R.F.P.S.Glasg., Lect. on Ment. Dis., Andr. Coll., Glasg.; 2, Woodside Crescent, Glasgow.

1912. Annandale, James Scott, M.B., Ch.B.Aberd., D.P.M., Medical Superintendent, Kingseat Mental Hospital, Newmachar, Aberdeen.

1904. Archdale, Mervyn Alexander, M.B., B.S.Durh., D.P.M., Medical Superintendent, Sunderland Mental Hospital, Ryhope.

1903. Armitage, Bernard William Francis, M.A., M.R.C.S., L.R.C.P., Assistant Physician, Bethlem Royal Hospital, Monk's Orchard, Eden Park, Beckenham, Kent.

1930. Armstrong, Robert William, B.Sc., M.D., B.Ch.Belf., D.P.M., Medical Superintendent, County and City Mental Hospital, Littlemore, Oxford.

1935. Arnott, Desmond William Henry, M.B., Ch.M., D.P.M.Sydney, Secretary of Section of Neurology and Psychiatry, B.M.A., N.S.W. Branch; "Harley", 143, Macquarie Street, Sydney, Australia.

1930. Ashby, William Ross, M.A., M.D., B.Ch.Cantab., M.R.C.S., L.R.C.P., D.P.M., Pathologist, St. Andrew's Hospital, Northampton.

1934. Aslett, Henry Edward Cullinan, M.R.C.S.Eng., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, Rauceby Mental Hospital, Sleaford, Lincs.

1927. Atkin, Isaac, M.D., M.R.C.S., L.R.C.P.Lond., D.P.M., Assistant Medical Officer, Park Prewett Mental Hospital, Basingstoke, Hants.

1936. Atlee, Charles Nelson, M.D.Dur., M.R.C.P.Lond., M.R.C.S.Eng., D.P.H., D.P.M., Barrister-at-Law, Divisional Medical Officer, L.C.C.; Visiting Medical Officer, Farmfield Certified Institution, Horley; 18, Sylvan Avenue, Finchley, N. 3.

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1922. Back, Frederick, M.B., B.S.Lond., M.R.C.S., L.R.C.P., D.P.M., Deputy Medical Superintendent, Sunderland Mental Hospital, Ryhope.
1926. Bailey, Josiah Reginald, M.B., Ch.B.Glasg., Medical Superintendent, Gogarburn Certified Institution, Corstorphine, Edinburgh.
1909. Bain, John, M.A., M.B., Ch.B.Glasg., Medical Superintendent, Derby Borough Mental Hospital, Rowditch.
1913. Bainbridge, Charles Frederick, M.B., Ch.B.Edin., Medical Superintendent, Devon County Mental Hospital, Exminster.
1906. Baird, Harvey, M.D., Ch.B.Edin., Medical Superintendent, Periteau House, Winchelsea, Sussex.
1934. Bamford, Charles Barry, M.D., Ch.B., D.P.M.Dubl., Medical Superintendent, Gateshead Mental Hospital, Stannington, Morpeth, Northumberland.
1922. Banbury, Percy, M.R.C.S., L.R.C.P., D.P.M., Medical Superintendent, Borough Mental Hospital, Ipswich.
1930. Barber, Leonard, M.D., B.S.Durh., L.R.C.P.&S.Edin., L.R.F.P.&S.Glasg., D.P.M., Assistant Medical Officer, City Mental Hospital, Fishponds, Bristol; Dunelm, Blackberry Hill, Fishponds, Bristol.
1934. Barbour, William Johnstone, M.D., Ch.B., D.P.M., Deputy Medical Superintendent, Borough Mental Hospital, Rowditch, Derby.
1922. Barclay, Rachel Mary, M.A., LL.B., M.D.Edin., Dipl. Psych., 2, W. Crosscauseway, Edinburgh.
1904. Barham, Guy Foster, M.A., M.D., B.Ch.Camb., M.R.C.S., L.R.C.P., Medical Superintendent, Claybury Hospital, Woodford Bridge, Essex.
1919. Barkas, Mary Rushton, M.Sc.N.Z., M.D., B.S., M.R.C.S., L.R.C.P., D.P.M., Taihoa, Tapu, Thames, New Zealand.
1927. Barkas, Thomas Cook, *O.B.E.*, M.B., B.S.Durh., Resident Medical Officer, Middleton Hall, Middleton St. George, co. Durham.
1923. Barnes, Francis Gregory Lawson, M.B., B.S.Lond., M.R.C.S., L.R.C.P., D.P.M., Medical Superintendent, Long Grove Hospital, Epsom, Surrey.
1935. Barnes, John Francis Lovel, M.A., M.R.C.S., L.R.C.P., Clinical Assistant, Department for Nervous Disorders, Radcliffe Infirmary, Oxford; Clinical Assistant, Oxford Educational Comm. Child Guidance Clinic; "Littlecote," Thame, Oxon.
1910. Bartlett, George Norton, M.B., B.S., M.R.C.S., L.R.C.P.Lond., "Brownie," Aldwick, Bognor Regis. (*Secretary, S.W Division*, 1916–22.)
1901. Baskin, J. Lougheed, M.D., L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Warming Hall, Aylesbury, Bucks.
1932. Bates, Ralph Marshall, F.R.C.S., D.P.M., Resident Medical Officer, Stoke Park Colony; Purdown House, Stapleton, Bristol.
1933. Beasley, Henry Eric, M.B., B.S., M.R.C.S., L.R.C.P., D.P.M., Resident Medical Officer, Middlesex Colony, Shenley.
1921. Beaton, Thomas, *O.B.E.*, M.D.Lond., F.R.C.P., Medical Superintendent, City Mental Hospital, Milton, Portsmouth. Lect. on Ment. Dis., Bethlem Royal Hosp. (*Assistant Editor*, 1926–27, *and Co-Editor of Journal*, 1927–31.)
1932. Beccle, Harold Charles, M.B., B.S., M.R.C.P., D.P.M., Assistant Medical Officer, Springfield Mental Hospital, S.W. 17.
1913. Bedford, Percy William Page, M.D., Ch.B., Dipl. Psych.Edin., Medical Superintendent, Dorset County Mental Hospital, Herrison, near Dorchester.
1934. Beggs, John, M.A., M.B., Ch.B., B.A.O.Dubl., Senior Assistant Physician, The Warneford, Oxford.
1922. Bell, Andrew Allan, M.B., Ch.B., F.R.F.P.S.Glasg., D.P.M., Medical Superintendent, Perth District Asylum, Murthly, Perthshire.
1934. Bell, Gavin John, M.B., B.S.Lond., D.P.M., Assistant Medical Officer, Cane Hill Hospital, Coulsdon, Surrey.
1931. Bell, George Macdonald, M.B., Ch.B.Edin., Medical Superintendent, Ladysbridge Mental Hospital, Banff.
1933. Benjacar, Edgar, M.D.Malta, Assistant Medical Officer, Royal Eastern Counties Institution, Colchester; The Turner Village, Turner Road, Mile End, Colchester.
1931. Bennet, Edward Armstrong, *M.C.*, M.A., M.D., D.P.M., Physician, The Institute of Medical Psychology, London; 97, Harley Street, W. 1.
1914. Bennett, James Wodderspoon, M.R.C.S., L.R.C.P.Lond., "Marsden", Babbacombe Road, Torquay, S. Devon.
1914. Benson, John Robinson, F.R.C.S.Eng., L.R.C.P.Lond., Resident Physician, Fiddington House, Market Lavington, Wilts, and Laverstock House, Salisbury. (*Chairman, S..W Division*, 1936–37.)

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1899. Beresford, Edwyn Henry, M.R.C.S., L.R.C.P., 20, Campion Road, Putney, S.W. 15.
1922. Berkeley-Hill, Owen Alfred Rowland, M.A., M.D., B.Ch.Oxon, M.R.C.S., L.R.C.P.Lond., Lt.-Col. *I.M.S.*, Station View, Ranchi, B.N. Railway, India.
1912. Berncastle, Herbert Melbourne, M.R.C.S., L.R.C.P., Toorak, 36, Birdhurst Rise, S. Croydon.
1927. Berry, Richard James Arthur, M.D.Edin.&Melb., F.R.C.S., F.R.S.Edin., Director of Medical Services, Stoke Park Colony, Stapleton, Bristol; "Rufford", Canford Lane, Westbury-on-Trym, Bristol.
1932. Bhathena, Sorab Jamasji, Lt.-Col. *I.M.S.* (retired), L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., L.M.Dubl., c/o Lloyds Bank, Ltd., Bombay, India.
1934. Biggart, John Henry, M.D., B.A.O., Pathologist to Scottish Mental Hospitals Laboratory, University Hall, St. Giles' House, Edinburgh.
1920. Birch, William Somerset, *M.C.*, M.R.C.S., L.R.C.P., Medical Superintendent, The Mental Hospital, Bridgetown; "Rosemont", Bridgetown, Barbados, British West Indies.
1894. Blachford, James Vincent, *C.B.E.*, M.D., B.S.Durh., M.R.C.S., L.R.C.P.Lond., M.P.C., Milverton House, Long Ashton, Somerset.
1935. Blacker, Carlos Paton, *M.C.*, M.A., D.M.Oxon., M.R.C.P., General Secretary, Eugenics Society, 69, Eccleston Square, S.W. 1.
1898. Blair, David, M.A., M.D., C.M.Glasg., 3a, Eton Terrace, Edinburgh.
1919. Blake, Stanley, L.R.C.P.&S.Irel., Deputy Resident Medical Superintendent, Portrane Mental Hospital, Donabate, co. Dublin.
1932. Blyth, William, M.D., Ch.B.Glasg., D.P.M., F.R.F.P.S., Senior Physician, Royal Scottish National Institution, Larbert, Stirlingshire.
1934. Bogle, Harriette Grenelle, M.D., Ch.B.Edin., D.P.M., Assistant Medical Officer, Holloway Sanatorium, Virginia Water, Surrey.
1900. Bolton, Joseph Shaw, D.Sc., M.D., B.S., F.R.C.P.Lond., Emeritus Professor of Mental Diseases, University of Leeds, "Merevale", Brownswood Road, Beaconsfield, Bucks. (PRESIDENT, 1928–29.)
1935. Boome, Edward James, M.B., Ch.B., M.R.C.P., D.P.H., T.D., Divisional Medical Officer, L.C.C., and Visiting Medical Officer, Farmfield Certified Institution; Greystacks, Hill View Road, Woking, Surrey.
1934. Booth, Edith Marion, M.B., Ch.B., Assistant Medical Officer, Bucks County Mental Hospital, Stone, near Aylesbury.
1922. Bostock, John, M.B., B.S., M.R.C.S., L.R.C.P., D.P.M., Lauriston, Wickham Terrace, Brisbane, Queensland, Australia.
1918. Bower, Cedric William, L.M.S.S.A., Physician-Superintendent, Springfield House, near Bedford.
1934. Boyd, Arthur Ross, M.B., B.Ch., B.A.O.R.U.I., Resident Medical Superintendent, County Mental Hospital, Antrim.
1926. Boyd, William, M.B., Ch.B.Edin., D.P.H., D.P.M., Medical Superintendent, Fife and Kinross District Asylum, Cupar, Fife.
1898. Boyle, A. Helen A., M.D.Brux., L.R.C.P.&S.Edin., 9, The Drive, Hove, Sussex.
1933. Boyle, Eric Patrick, M.B., Ch.B.Edin., D.P.M.Eng., Green Orchard, Powick, near Worcester.
1926. Braithwaite, Joseph, M.B., Ch.B.Edin., D.P.M., Assistant Medical Officer, Cumberland and Westmorland Mental Hospital, Garlands, Carlisle.
1922. Bramwell, Edwin, M.D., F.R.C.P.Edin.&Lond., F.R.S.Edin., Physician, Royal Infirmary, and Prof. of Clin. Med., Univ. of Edin., 23, Drumsheugh Gardens, Edinburgh.
1911. Brander, John, M.D., Ch.B.Edin., F.R.C.P., D.P.M., Medical Superintendent, Friern Hospital, New Southgate, N. 11.
1925. Brennan, Richard Dominick, M.B., B.Ch.N.U.I., D.P.M., Assistant Medical Officer, District Mental Hospital, Waterford.
1930. Bristowe, Hubert C., M.D.Lond., M.R.C.S., L.R.C.P., M.P.C., Hon. Major *R.A.M.C.* (retired), 3, Upper Belgrave Road, Clifton, Bristol.
1924. Brown, Basil William, M.B., B.S.Lond., L.M.S.S.A., D.P.M., The Priory, Roehampton, London, S.W. 15.
1905. Brown, Harry Egerton, *O.B.E.*, M.D., Ch.B.Glasg., M.P.C., 116, Hope Street, Glasgow.
1908. Brown, Robert Dods, M.D., Ch.B., F.R.C.P.Edin., D.P.H., Dipl. Psych., Physician-Superintendent, Royal Mental Hospital, Aberdeen.
1935. Brown, Roger Crosbie, M.R.C.S., L.R.C.P., Medical Officer, H.M. Prison, Liverpool.
1916. Brown, William, D.Sc.Lond., M.A., M.D., B.Ch.Oxon., F.R.C.P., Wilde Reader in Mental Philosophy, Univ. Oxford, 88 Harley Street, London, W. 1.

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1935. Brownlees, Thomas John Kilpatrick, L.R.C.P., M.R.C.S., Assistant Medical Officer, Devon Mental Hospital, Exminster, near Exeter.
1917. Bruce, Alexander Ninian, D.Sc., M.D., F.R.C.P.Edin., Lect. on Neurol., Univ. of Edin.; 8, Ainslie Place, Edinburgh.
1932. Bruce, David Charles, M.B., Ch.B.Edin., Dipl. Psych. Edin., Medical Superintendent, Criminal Lunatic Department and State Institution for Defectives, Perth.
1928. Bruce, David Kennedy, M.B., Ch.B.Glasg., D.P.M., Senior Assistant Medical Officer, Storthes Hall Mental Hospital, Kirkburton, near Huddersfield.
1893. Bruce, Lewis C., *M.C.*, M.D., F.R.C.P.Edin., M.P.C., Park End, Lockerbie, Dumfriesshire, (*Secretary, Scottish Division*, 1901–1907; *Co-Editor of Journal*, 1911–16; *Chairman, Scottish Division*, 1934–35.)
1927. Brunlees, Oswald, L.R.C.P.&S.Edin., 75, Denmark Villas, Hove, Sussex.
1913. Brunton, George Llewellyn, M.D., Ch.B.Edin., Medical Superintendent, City Mental Hospital, Mapperley Hill, Nottingham.
1929. Bryce, William Henderson, M.B., C.M.Edin., Gifford Bank, Gifford, East Lothian.
1928. Burke, Noel Hawley Michael, M.R.C.S., L.R.C.P., D.P.M., D.M.R.E.Camb., Medical Superintendent, Cell Barnes Colony, St. Albans, Herts.
1932. Burns, Robert Henderson, B.A.Cantab., M.R.C.S., L.R.C.P., Assistant Medical Officer, Claybury Hospital, Woodford Bridge, Essex.
1936. Barrows, Thomas Eggleston, B.A.Cantab., M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Burghill, Hereford.
1935. Burt, Hutchinson, M.B., Ch.B., D.P.M.Leeds, Assistant Medical Officer, West Riding Mental Hospital, Wakefield.
1924. Bushe, Charles Kendal, *O.B.E.*, B.A., M.D., B.Ch.Dubl., Surg.-Rear-Admiral R.N., (retired), Hill End Hospital, St. Albans, Herts.
1921. Buzzard, Sir Edward Farquhar, Bart., *K.C.V.O.*, M.A., M.D., B.Ch.Oxon., F.R.C.P., Regius Professor of Medicine, Oxford University; 85, Banbury Road, Oxford.

1928. Calder, Flora Hannah Macdonald, M.A., M.D., D.P.H.Edin., Commissioner, Board of Control, Metropole Buildings, Northumberland Avenue, W.C. 2.
1921. Caldicott, Charles Holt, *M.B.E.*, M.B., M.R.C.S., L.R.C.P.Lond., Grantbourne, Chobham, Surrey.
1928. Caldwell, William Alexander, M.R.C.P., M.R.C.S., D.P.M., Assistant Medical Officer, Long Grove Hospital, Epsom.
1925. Cameron, Donald Ewen, M.B., Ch.B.Glasg., D.P.M., Worcester State Hospital, Worcester, Mass., U.S.A.
1936. Cameron, Kenneth, M.B., Ch.B., Dipl. Psych., Assistant Physician, Edinburgh Royal Mental Hospital, Morningside, Edinburgh.
1936. Campbell, Margaret Catherine, M.B., Ch.B.Glas., Assistant Medical Officer, West Riding Mental Hospital, Wakefield.
1897. Campbell, Robert Brown, M.D., C.M., F.R.C.P.Edin., 15, Douglas Crescent, Edinburgh 12. (*Secretary, Scottish Division*, 1910–20, *and Chairman*, 1929–30; PRESIDENT, 1932–33.)
1932. Cannon, Alexander, M.A., M.D., Ph.D., D.P.M., 152, Harley Street, London, W. 1.
1930. Cannon, H. C. A. Tandy, Surgeon Lt.-Commander *R.N.* (Ret.), L.R.C.P.&S.I., Resident Physician, Moorcroft House, Hillingdon, Middlesex.
1936. Cant, John Arthur Pinney, M.B., Ch.B.Bir., Assistant Medical Officer, Coleshill Hall, Coleshill, Birmingham.
1927. Carey, Catherine, M.B., B.Ch.N.U.I., Assistant Medical Officer, District Mental Hospital, Cork.
1932. Carroll, Denis Charles, M.A.Camb., M.R.C.S., L.R.C.P., 22, Queen Anne Street, W. 1.
1933. Carruthers, Peter Walter, M.B., Ch.B.Edin., Assistant Medical Officer, The Old Manor, Salisbury.
1935. Carse, Joshua, M.D., D.P.M., Deputy Medical Superintendent, County Mental Hospital, Berry Wood, Northampton.
1928. Cassin, Patrick Joseph, M.D., B.Ch.N.U.I., D.M.D., Resident Medical Superintendent, Mental Hospital, Kilkenny, Ireland.
1922. Casson, Elizabeth, M.D., Ch.B.Brist., D.P.M., Dorset House, Clifton, Bristol 8.
1934. Cates, Bernard, M.R.C.S., L.R.C.P., D.P.M.Lond., 11, Wentworth Road, Golders Green, N.W. 11.
1928. Cates, Henry Joseph, M.D.Lond., D.P.H., Medical Superintendent, Northwoods House, Winterbourne, Bristol.
1932. Chalmers, Edith, M.B., Ch.B.Glasg., Assistant Medical Officer, Royal Scottish National Institution, Larbert, Stirlingshire.
1911. Chambers, Walter Duncanon, M.A., M.D., Ch.B., F.R.C.P.Edin., M.P.C., Physician Superintendent, James Murray's Royal Asylum; Murray House, Perth.

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1928. Chapman, Theonie Renée, M.B., B.S.Durh., Assistant Medical Officer, Northumberland Mental Hospital, Morpeth.
1932. Chapman, William Noel James, M.B., Ch.B., D.P.H., Dipl. Psych. Edin., Medical Superintendent, Lanark District Asylum, Hartwood.
1928. Chennell, Eileen Anne, M.B., B.S., M.R.C.S., L.R.C.P., D.P.M., Brooke House, Clapton, E. 5.
1923. Chevens, Leslie Charles Frederick, M.R.C.S., L.R.C.P., D.P.M., 827, Sidcup Road, New Eltham, S.E. 9.
1917. Chisholm, Percy, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Medical Superintendent, Queen Mary Hospital, Hanmer Springs, New Zealand.
1907. Chislett, Charles Game Angus, M.B., Ch.B., F.R.F.P.S Glasg., Medical Superintendent, Stoneyetts, Chryston, Lanark.
1921. Cholmeley, Montague Adye, M.R.C.S., L.R.C.P.Lond., D.P.M. (retired), 43, Upper Richmond Road, London, S.W. 15.
1907. Clarke, Geoffrey, M.D.Lond., Medical Superintendent, Bexley Hospital (L.C.C.), Bexley, Kent.
1907. Clarkson, Robert Durward, B.Sc., M.D., C.M., F.R.C.P.Edin., Lect. on Ment. Deficiency, Univ. of Edin.; Little Cambus, Doune, Perthshire. (*Chairman, Scottish Division*, 1935–36.)
1931. Clegg, John Leonard, M.B., Ch.B., D.P.H., D.P.M., Assistant Medical Officer, South Yorkshire Mental Hospital, Sheffield.
1934. Clifford, Louis Seymour, L.R.C.P.I.&L.M., L.R.C.S.I.&L.M., Assistant Medical Officer, Criminal Lunatic Asylum, Dundrum, co. Dublin.
1925. Cobb, Geoffrey Francis, M.R.C.S., L.R.C.P., D.P.M., M.P.C., Eghurst Court, Kingswood, Surrey.
1933. Colahan, Arthur Nicholas Whistler, M.B., Ch.B., 12, De Montfort Street, Leicester.
1900. Cole, Sydney John, M.A., M.D., B.Ch.Oxon., Alphen, Sea Lane, Goring-by-Sea, Worthing, Sussex.
1930. Coleman, Stanley Maurice, M.R.C.S., L.R.C.P., D.P.M., Deputy Medical Superintendent, Runwell Hospital, near Wickford, Essex.
1906. Collier, Walter Edgar, M.R.C.S., L.R.C.P.Lond., Ailsa Cottage, Parkfield, Seal, Sevenoaks, Kent.
1903. Collins, Michael Abdy, *O.B.E.*, M.D., B.S., M.R.C.S., L.R.C.P.Lond., Ministry of Pensions. Medical Services Division; Burwood House, Whitstable Road, Canterbury. (*General Secretary*, 1912–18; *Vice-Chairman, Educational Committee*, 1919–27; *Chairman, S.E. Division*, 1932–33; PRESIDENT, 1936–37.)
1934. Conlon, Thomas Joseph, L.R.C.P.I.&L.M., L.R.C.S.I.&L.M., D.P.M., Assistant Medical Officer, District Mental Hospital, Enniscorthy, co. Wexford.
1910. Conlon, Thomas Peter, L.R.C.P.&S.Irel., Resident Medical Superintendent, District Mental Hospital, Monaghan.
1921. Connell, Ernest Henry, M.B., Ch.B.Edin., D.P.M., 9, Redford Crescent, Colinton, Edinburgh.
1920. Connell, Oliver George, *M.C.*, L.R.C.P.&S.Irel., Medical Superintendent, St. Andrew's Hospital, Thorpe, Norwich.
1914. Connolly, Victor Lindley, *M.C.*, M.B., B.Ch.Belf., D.P.M., Medical Superintendent, Hants County Mental Hospital, Park Prewett, Basingstoke.
1927. Cook, Leslie Colin, M.R.C.S., L.R.C.P., D.P.M., Deputy Medical Superintendent, Bexley Hospital (L.C.C.), Bexley, Kent.
1930. Cooper, Hugh Astley, M.B., B.S.Lond., M.R.C.P., D.P.M., Deputy Medical Superintendent, Hants County Mental Hospital, Knowle, Fareham.
1927. Cooper, James Woodman Astley, L.S.A., L.R.C.S., L.R.C.P., Medical Superintendent, Middleton Hall Private Mental Hospital, and Almora Hall, Middleton St. George, Durham.
1928. Copeland, Cecil Leonard, M.B., Ch.B.Liverp., D.P.M., Deputy Medical Superintendent, Cefn Coed Hospital, Swansea.
1903. Cormac, Harry Dove, M.B., M.S.Madras, D.P.M., Medical Superintendent, Cheshire County Mental Hospital, Parkside, Macclesfield; Lect. on Ment. Dis., Univ. of Manch.; Parkside House, Macclesfield. (*Chairman, N. and M. Division*, 1931–32.)
1891. Corner, Harry, M.D., M.R.C.S., L.R.C.P., M.P.C., Jersey Farm, Sandridge, near St. Albans, Herts.
1910. Coupland, William Henry, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Flat 8, Manor Court, Leigham Avenue, High Road, Streatham, S.W. 16.
1932. Courtney, Patrick J., M.D.N.U.I., Assistant Medical Officer, Donegal Mental Hospital Letterkenny.

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1931. Couston, Alastair Gordon, L.R.C.P.&S.Edin., Sunnyside Mental Hospital, Christchurch, New Zealand.
1936. Cox, Alice, M.B., Ch.B.Glasg., Mental Specialist to Devon County Council Education and Mental Deficiency Committees; 4, Bamfield Crescent, Exeter.
1911. Cox, Donald Maxwell, M.R.C.S., L.R.C.P.Lond., Deputy Medical Superintendent, Hereford County Mental Hospital, Burghill.
1926. Coyne, William Joseph, M.D., B.Ch.N.U.I., D.P.M., Resident Physician, Mountjoy Civil Prison, Dublin, N.W. 4.
1924. Craig, Roy Neville, M.D.Durh., M.R.C.S., L.R.C.P.Lond., D.P.M., 23, Dix's Field, Exeter.
1911. Crichlow, Charles Adolphus, M.B., Ch.B.Glasg., Senior Assistant Medical Officer, Bangour Village, Uphall, Linlithgowshire.
1930. Crichton-Miller, Hugh, M.A., M.D.Edin., M.D.Pavia, Hon. Senior Physician, The Institute of Medical Psychology; 6, Devonshire Place, W. 1.
1915. Crosthwaite, Frederick Douglas, M.B., Ch.B.Edin., D.P.H., Physician Superintendent, Witrand Institute for Feeble-minded, Potchefstroom, Transvaal, S. Africa.
1923. Crow, Norah Annie, M.A.Edin., M.D., B.S.Lond., "Kingsclere", Harrington Road, Brighton.
1935. Crowley, Timothy, M.B., Ch.B.Edin., Assistant Medical Officer, Caterham Hospital (L.C.C.), Caterham, Surrey.
1933. Curran, Desmond, M.B., B.Ch.Camb., M.R.C.P., D.P.M., Physician and Lect. on Psych. Med., St. George's Hospital; 14, Wimpole Street, W. 1.
1933. Curran, James Thomas, M.B., Ch.B.Glasg., Deputy Medical Superintendent, Stoneyetts Certified Institution, Chryston, Lanarkshire.
1935. Curran, Thomas Patrick, M.B., Ch.B., B.A.O., D.P.M., Deputy Medical Superintendent, County Mental Hospital, Prestwich, near Manchester.
1919. Cuthbert, James Harvey, M.B., Ch.B.Edin., D.P.M., Medical Superintendent, West Ham Mental Hospital, Goodmayes; "The Leazes", Barley Lane, Goodmayes, Essex.
1936. Cuthbert, Theodore Martin, M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Kent County Mental Hospital, Barming Heath, Maidstone.
1930. Cutts, George Lambert, M.R.C.S., L.R.C.P.Lond., L.D.S., D.P.M., Deputy Medical Superintendent, Friern Hospital, New Southgate, N. 11.

1934. Daly, Patricia Mary Josephine, M.B., B.Ch., B.A.O.N.U.I., D.P.H., Assistant Medical Officer, Portrane Mental Hospital, Donabate, co. Dublin.
1935. Danby, Thomas Anyan, M.B., Ch.B.Vict., D.P.H., Capt. R.A.M.C.(T.A.), Medical Superintendent, Sandhill Park, Bishop's Lydeard, Taunton; "Wayside", Bishop's Lydeard, Taunton.
1907. Daniel, Alfred Wilson, B.A., M.D., B.Ch.Camb., M.R.C.S., L.R.C.P.Lond., Red Roofs, 25, Lauderdale Drive, Petersham, Surrey. (*Secretary, Educational Committee*, 1920–27.)
1926. Darlington, Arthur, B.A., M.B., B.Ch.Dubl., D.P.H., D.P.M., Deputy Medical Superintendent, Somerset and Bath Mental Hospital, Wells.
1930. Das, Banarsi, M.B., B.S.Punjab, D.P.M., Superintendent, Mental Hospital, Agra, India.
1896. Davidson, Andrew, M.D., C.M.Aberd., M.P.C., 221, Macquarie Street, Sydney, New South Wales.
1925. Davidson, Thomas Wishart, M.B., Ch.B.Glasg., D.P.M., Deputy Medical Superintendent and Pathologist, City Mental Hospital, Humberstone, Leicester.
1922. Davie, Thomas Macnaughton, *M.C.*, M.D., Ch.B.Edin., Ph.D., D.P.M., Barrister-at-Law, Medical Superintendent, East Riding Mental Hospital, Beverley, Yorks.
1935. Davies, Isaac John, M.B., B.Ch., B.Sc.Wales, D.P.M., Senior Assistant Medical Officer, Cardiff City Mental Hospital, Whitchurch, Glam.
1931. Davies, Sidney, M.B., B.S.Lond., L.M.S.S.A., D.P.M., Medical Superintendent, Joint Counties Mental Hospital, Carmarthen.
1928. Davies, Stuart Wyndham, M.R.C.S., L.R.C.P.Lond., D.P.M., Assistant Medical Officer, Hensol Castle, nr. Pontyclun, Glam.
1921. Davies-Jones, Charles William Saunderson, M.B., Ch.B.Edin., Medical Superintendent, Isle of Wight Mental Hospital, Whitecroft, Newport.
1920. Dawson, William Siegfried, M.A., M.D., B.Ch.Oxon., F.R.C.P., D.P.M., Professor of Psychiatry, University of Sydney; B.M.A. House, 135, Macquarie Street, Sydney, New South Wales, Australia.
1926. Deane, Frederick John, M.B., Ch.B.Edin., D.P.M., 24, Cyprus Avenue, Belfast.
1935. Dedman, James Baillie, M.B., Ch.B.Edin., D.P.M., Assistant Medical Officer, Cornwall Mental Hospital, Bodmin, Cornwall.

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1925. Delany, John James, L.R.C.P.&S.Irel., D.P.M., 68, Wellington Road, Dublin.
1929. Denyssen, John Abraham Felix, M.R.C.S., L.R.C.P.Lond., Assistant Medical Officer, Banstead Hospital (L.C.C.), Sutton, Surrey.
1901. De Steiger, Adele Isabella, M.D.Lond., 36, Clarendon Road, London, W. 11.
1905. Devine, Henry, *O.B.E.*, M.D., B.S., F.R.C.P.Lond., M.R.C.S.Eng., M.P.C., Medical Superintendent, Holloway Sanatorium, Virginia Water, Surrey; The Ridge, Virginia Water, Surrey. (*Assistant Editor*, 1916–1920, *and Co-Editor of Journal*, 1920–27.)
1936. De Vine, Michael George, M.B., B.Ch.N.U.I., 126, Harley Street, W. 1.
1904. Devon, James, L.R.C.P.&S.Edin., F.R.F.P.S.Glasg., 67, Great King Street, Edinburgh.
1932. Dewar, Douglas Campbell, M.B., Ch.B.Glasg., L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., D.P.M.Lond., Assistant Medical Superintendent, Renfrew District Asylum, Dykebar, Paisley.
1925. Dhunjibhoy, Jal Edulji, M.B., B.S.Bomb., Major *I.M.S.*, Medical Superintendent, The Indian Mental Hospital, Kanké, Ranchi, Bihar and Orissa, India; c/o Lloyds Bank, 6, Pall Mall, S.W. 1.
1921. Dick, Alexander, *M.C.*, M.B., Ch.B.Glasg., Deputy Medical Superintendent, Glasgow District Mental Hospital, Woodilee, Lenzie.
1922. Dickson, James, *M.C.*, M.B., Ch.B.Edin., "Whitehill", The Broadway, Thorpe Bay.
1936. Dickson, Robert F. G., L.R.C.P.&S.I., D.P.H., Senior Assistant Medical Officer, Down County Mental Hospital, Downpatrick, Ireland.
1915. Dillon, Frederick, M.D., Ch.B.Edin., Medical Superintendent, Northumberland House, Finsbury Park, London, N. 4.
1909. Dillon, Kathleen, L.R.C.P.&S.Irel., Assistant Medical Officer, District Asylum, Mullingar.
1905. Dixon, J. Francis, M.A., M.D., B.Ch.Dubl., M.P.C., Medical Superintendent, City Mental Hospital, Humberstone, Leicester.
1879. Dodds, William John, D.Sc., M.D., C.M.Edin., 19, Marina Road, Prestwick, Ayrshire.
1892. Donelan, John O'Conor, L.R.C.P.&S.Irel., M.P.C., Medical Superintendent, Grangegorman Mental Hospital; Lect. on Ment. Dis., Univ. of Dubl., St. Dymphna's, North Circular Road, Dublin. (*Chairman, Irish Division*, 1931–33.)
1928. Douglas-Morris, Ernest Mannering, L.M.S.S.A., Neurologist, Ministry of Pensions Clinic, Nottingham; Tykeford Abbey Nursing Home, Newport Pagnell, Bucks.
1919. Drake-Brockman, Henry George, M.R.C.S., L.R.C.P.Lond., Medical Superintendent, St. Luke's Hospital, Middlesbrough.
1933. Drummond, Peter, M.B., Ch.B., Medical Superintendent, Mid-Wales Counties' Mental Hospital; Chancefield, Talgarth, Breconshire.
1916. Drummond, William Blackley, M.D., C.M., F.R.C.P.Edin., Garth Hill, N. Queensferry, Fife.
1921. Drury, Kenneth Kirkpatrick, *M.C.*, B.A., M.D., B.Ch.Dubl., D.P.M., Medical Superintendent, Leicester and Rutland Mental Hospital, Narborough, near Leicester. (*Secretary, Educational Committee* since 1936.)
1907. Dryden, Arthur Mitchell, M.B., Ch.B.Edin., Medical Superintendent, Glasgow District Mental Hospital, Gartloch, Gartcosh.
1899. Dudley, Francis, L.R.C.P.&S.Irel., "Hilton", Keyberry Park, Newton Abbott, Devon.
1926. Duncan, Archibald Glen, M.D., B.S., M.R.C.S., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, Essex County Mental Hospital, Severalls, Colchester.
1936. Duncan, Jessie Galloway, M.B., Ch.B.Glasg., D.P.H.Camb., Divisional Medical Officer, L.C.C.; Visiting Medical Officer, South Side Home, Streatham; 1, Steep Hill, London, S.W. 16.
1923. Dunne, John, M.B., B.Ch.R.U.I., D.M.D., Resident Medical Superintendent, District Mental Hospital, Sligo.
1932. Dymock, Thomas, M.D., Ch.B.Glasg., Assistant Medical Officer, Edinburgh District Mental Hospital, Bangour, West Lothian.

1906. Eager, Richard, *O.B.E.*, M.D., Ch.B.Aberd., M.P.C., Crossways, Countess Weir, near Exeter.
1931. Earl, Charles James Cecil, F.R.C.P.I., D.P.M., Assistant Medical Officer, Caterham Hospital (L.C.C.), Caterham, Surrey.
1891. Earls, James Henry, M.D., M.Ch.R.U.I., L.S.A., D.P.H., M.P.C., Barrister-at-Law; 5, Becmead Avenue, Streatham, London, S.W. 16.
1921. East, Guy Rowland, M.D., B.S., B.Hy.Durh., D.P.H., Medical Superintendent, Northumberland County Mental Hospital, Cottingwood, Morpeth.
1907. East, Wm. Norwood, M.D.Lond., F.R.C.P., M.P.C., One of His Majesty's Commissioners of Prisons; Prison Commission, Home Office, Whitehall, S.W. 1.

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1895. Easterbrook, Charles C., M.A., M.D., F.R.C.P.Edin., M.P.C., ***J.P.***, Physician-Superintendent, Crichton Royal Institution, Dumfries.
1931. Eaves, Elizabeth Cowper, M.D., B.S., D.P.M.Lond., Honorary Neuro-pathologist, South Yorkshire Mental Hospital, Wadsley, Sheffield; Lect. on Physiol., Univ. of Sheffield.
1924. Eddison, Herbert Wilfred, M.A., M.D.Camb., M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Wonford House Hospital, Exeter.
1895. Edgerley, Samuel, M.A., M.D., C.M.Edin., M.P.C., Holly Bank, Menston, Leeds. (***Chairman, Northern and Midland Division***, 1934–36.)
1897. Edwards, Francis Henry, M.D.Brux., M.R.C.P.Lond., " Cherchefelle ", Reigate, Surrey.
1924. Edwards, Thomas Lloyd, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., D.P.M., Assistant Medical Officer, Glamorgan County Mental Hospital, Bridgend.
1919. Eggleston, Henry, M.B., B.S.Durh., M.P.C., c/o Standard Bank of S. Africa, 10, Clement's Lane, E.C. 4.
1901. Elgee, Samuel Charles, *O.B.E.*, L.R.C.P.&S.Irel., Clougheast, The Gorses, Cooden, Bexhill-on-Sea.
1936. Elphinstone, John Henry, M.R.C.S.Eng., L.R.C.P., L.M.S.S.A.Lond., Civil Medical Practitioner to the Military Hospital, Shorncliffe; 2, Cherry Garden Avenue, Folkestone, Kent.
1901. Erskine, Wm. J. Adams, M.D., C.M.Edin., The Orchard, Ravenmeols Lane, Formby, near Liverpool.
1925. Esson, Walter Louis, M.A., M.B., Ch.B.Aberd., 21, Oldham Road, Miles Platting, Manchester.
1895. Eurich, Frederick Wilhelm, M.D., C.M.Edin., Lanshawe Cottage, Ilkley, Yorks. Prof. of For. Med., Univ. of Leeds.
1934. Eustace, Henry Jocelyn, M.B., B.Ch., B.A.O.Dubl., Assistant Medical Officer, Hampstead, Glasnevin, Dublin.
1909. Eustace, William Neilson, L.R.C.P.&S.Irel., Lisronagh, Glasnevin, Dublin.
1918. Evans, Albert Edward, M.B., B.S.Lond., M.R.C.S., L.R.C.P.Lond., D.P.H., Commissioner, Board of Control; 11, Weymouth Avenue, Mill Hill, N.W. 7.
1927. Ewan, Grey Lamont, *J.P.*, B.Sc., M.B., Ch.M.Sydney, D.P.M., Medical Superintendent, The Mental Hospital, Stockton, nr. Newcastle, New South Wales.
1930. Ewen, John Harold, M.R.C.P.E., D.P.M., Deputy Medical Superintendent, Surrey County Mental Hospital, Netherne, Coulsdon.
1914. Ewing, Cecil Wilmot, L.R.C.P.&S.Irel., D.P.M., Medical Superintendent, Storthes Hall Mental Hospital, Kirkburton, nr. Huddersfield.

1929. Fairbairn, William Ronald Dodds, M.A., M.D., Ch.B.Edin., Dipl. Psych., Psychologist, Jordanburn Nerve Hospital; 18, Lansdowne Crescent, Edinburgh.
1925. Fairweather, Anne, M.D., B.S.Durh., D.P.M., The Red House, Haydon Bridge, Northumberland.
1933. Faull, John Langdon, M.R.C.S., L.R.C.P., D.P.M., M.R.C.P.Lond.,
1908. Fenton, Henry Felix, M.B., Ch.B.Edin., Medical Superintendent, Worcester County and City Mental Hospital, Powick.
1930. Fenwick, Philip Cuthbert Collingwood, L.M.S.S.A., Deputy Medical Superintendent, East Sussex Mental Hospital, Hellingly.
1928. Finiefs, Leonidas Aristodimos, M.R.C.S., L.R.C.P.Lond., M.D.Paris, Deputy Medical Superintendent, Three Counties Mental Hospital, Arlesey, Beds.
1889. Finlay, David, M.D., C.M.Glasg. (retired), Parkhurst, Park Street, Bridgend, Glamorgan.
1906. Firth, Arthur Harcus, M.A., M.D., B.Ch.Edin., Medical Superintendent, Worcester County Mental Hospital, Barnsley Hall, Bromsgrove.
1930. Fisher, John William, M.R.C.S., L.R.C.P., D.P.H., D.P.M., Assistant Medical Officer, Devon Mental Hospital, Exminster, S. Devon; Glen Lea, 58, Oldway Road, Paignton, S. Devon.
1903. Fitzgerald, Alexis, L.R.C.P.&S.Irel., Medical Superintendent, District Mental Hospital, Waterford.
1929. FitzGerald, Edward Joseph, M.D., B.Ch.N.U.I., Medical Superintendent, Bridge Home, Witham, Essex.
1908. Fitzgerald, James Francis, L.R.C.P.&S.Irel., Resident Medical Superintendent, District Mental Hospital, Clonmel, co. Tipperary.
1928. Fitzgerald, John, M.D., B.Sc.N.U.I., D.P.M., D.P.H., Resident Medical Superintendent, Mental Hospital, Mullingar.
1930. Fitzgerald, John Joseph, M.D.Durh.&Brux., Glengariff, Crosthwaite Park, Kingstown, co. Dublin.

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1930. Fitzgerald, Nora May, M.B., B.Ch., B.A.O., Mental Hospital, Mullingar.
1933. Fitzgerald, Otho William Strangman, M.B., B.Ch.Dubl., D.P.M., Assistant Medical Officer, Shenley Mental Hospital, Shenley, Herts.
1921. Fleming, Gerald William Thomas Hunter, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Hereford County and City Mental Hospital, Burghill, Hereford. (*Assistant Editor*, 1931–32, *and Co-Editor of Journal since* 1932.)
1904. Fleming, Wilfrid Louis Remi, M.R.C.S., L.R.C.P.Lond., *J.P.*, Suffolk House, Pirbright, Surrey.
1925. Flind, James, M.D., Ch.B.Glasg., D.P.M., Deputy Medical Superintendent, Peckham House, London, S.E. 15.
1930. Forbes, Donald John, M.B., Ch.B.St.And., Medical Superintendent, Baldovan Certified Institution, Craigmill House, by Dundee.
1927. Ford-Robertson, William Marsden, M.D., Ch.B.Edin., Director, West of Scotland Neuro-Psychiatric Research Institute; 28, Hamilton Park Avenue, Glasgow, W. 2.
1926. Forrester, Robert Cairns, M.B., Ch.B.Edin., D.P.M., Assistant Medical Officer, City of London Mental Hospital, Stone, Dartford.
1916. Forsyth, Charles Wesley, M.D., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Winson Green Mental Hospital, Birmingham; Clin. Lect. on Ment. Dis., Univ. of Birmingham.
1913. Forward, Ernest Lionel, M.R.C.S., L.R.C.P.Lond., D.C.M.S., Little Forton, Gerrard's Cross, Bucks.
1925. Fox, Francis Elliot, B.A.Camb., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Brislington House, Bristol.
1920. Fox, J. Tylor, M.A., M.D., B.Ch.Camb., M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Lingfield Epileptic Colony; The Homestead, Lingfield, Surrey.
1923. Franklin, Marjorie Ellen, M.B., B.S., M.R.C.S., L.R.C.P.Lond., D.P.M., Physician, London Clinic for Psycho-Analysis; 81, Wimpole Street, London, W. 1.
1919. Fraser, Kate, B.Sc., M.D., Ch.B.Glasg., D.P.H., Deputy Commissioner, General Board of Control, Scotland; 25, Palmerston Place, Edinburgh.
1928. Fraser, William, B.Sc., M.B., Ch.B.Glasg., D.P.H., D.P.M., Deputy Medical Superintendent, North Riding Mental Hospital, York.
1935. French, Margaret, M.B., B.S., Medical Officer, Chiswick House, Pinner.
1936. Frew, John McIntyre, M.B., Ch.B.Glas., D.P.M., Assistant Medical Officer, North Riding Mental Hospital, York.
1921. Fuller, Hugh Hercus Cavendish, M.D., Ch.B.Edin., 40, Harley Street, London, W. 1.

1928. Gall, Herbert, M.R.C.S., L.R.C.P.Lond., Lt.-Col., *R.A.M.C.*, 250, St. James's Court, London, S.W. 1.
1928. Gallagher, Charles Eddie, M.R.C.S., L.R.C.P.Lond., "Killiecrankie", 196, Main Road, Sidcup, Kent.
1932. Galloway, James Forbes, M.D.Liverp., D.P.H., Public Health Offices, Wood Street, Doncaster.
1928. Gardner, Dorothy Margaret, M.B., B.Ch.Belf., D.P.H., Assistant Medical Officer, Belfast Mental Hospital, Purdysburn, Belfast.
1931. Garrod, Marjorie, M.R.C.S., L.R.C.P., Hon. Medical Registrar, The Institute of Medical Psychology; "Bankcroft", Douglas Road, Harpenden, Herts.
1912. Garry, John William, M.B., B.Ch.N.U.I., Assistant Medical Superintendent, District Mental Hospital, Ennis, co. Clare.
1896. Geddes, John William, M.B., C.M.Edin., 4, Ambrose House, Worthing.
1930. Gemmell, Daniel Topping, L.R.C.P.&S.I., L.R.T.P.&S.G., D.P.M., Dornhurst, Halstead, Essex.
1919. Gifford, John, B.A.Cape, M.B., Ch.B.Edin., D.P.M., Medical Superintendent, County Mental Hospital, Prestwich.
1931. Gilchrist, Thomas, L.R.C.P.&S.Edin., L.R.C.P.S.Glasg., Assistant Medical Officer, Park Prewett Mental Hospital, Basingstoke.
1931. Gill, Samuel Ernest, M.D.Lond., D.P.H., 8, Grand Parade, Brighton.
1923. Gillespie, Isabella Anne, M.B., B.Ch.Edin., D.P.M., Deputy Medical Superintendent, Cheshire County Mental Hospital, Upton, Chester.
1921. Gillespie, Robert Dick, M.D., Ch.B.Glasg., F.R.C.P.Lond., D.P.M., Physician and Lect. on Psych. Med., Guy's Hosp., 16, Chester Terrace, Regent's Park, N.W. 1.
1920. Gillis, Kurt, M.B., Ch.B.Edin., Witrand Institution, Potchefstroom, Transvaal, S. Africa.

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1897. Gilmour, John Rutherford, M.B., C.M., F.R.C.P.Edin., M.P.C., Medical Superintendent, West Riding Mental Hospital, Scalebor Park, Burley-in-Wharfedale, Yorks. (*Secretary, N. and M. Division*, 1920–28, *and Chairman*, 1929–31.)

1906. Gilmour, Richard Withers, M.B., B.S.Durh., M.R.C.S., L.R.C.P.Lond., Wilsley House, Holme Lacy, Hereford.

1929. Glaister, John Norman, M.B., B.S., M.R.C.S., L.R.C.P.Lond., Deputy Medical Superintendent, Northumberland House; Physician, British Hospital for Nervous and Mental Disorders; 2, Devonshire Place, London, W. 1.

1936. Glass, Joseph Vincent, M.B., B.Ch., B.A.O.N.U.I., D.P.M., Assistant Medical Officer, District Mental Hospital, Carlow, Ireland.

1923. Golla, Frederick Lucien, M.A., M.B., B.Ch.Oxon., F.R.C.P.Lond., Director of the Laboratory and Pathologist, London County Mental Hospitals, The Maudsley Hospital, Denmark Hill, London, S.E. 5; Professor of the Pathology of Mental Diseases, University of London; The Dene, Sunninghill, Berks. (*Chairman, Research and Clinical Committee since* 1931.)

1897. Good, Thomas Saxty, *O.B.E.*, M.A.Oxon., M.R.C.S., L.R.C.P.Lond., 21, Carlisle Mansions, Carlisle Place, S.W. 1. Lect. on Psychiat., Univ. of Oxford. (PRESIDENT, 1930–31.)

1889. Goodall, Edwin, *C.B.E.*, M.D., B.S., F.R.C.P.Lond., M.P.C., "Fairlawn", Kingsway, Hove, Sussex. (*Assistant Editor*, 1894–5, *and Co-Editor of Journal*, 1895–98; PRESIDENT, 1923–24.)

1920. Gordon, George, M.B., B.Ch.Glasg., 59, Queen Anne Street, W. 1.

1928. Gordon, Ronald Grey, D.Sc., M.D., F.R.C.P.Edin., 2, Catherine Place, Bath.

1901. Gostwyck, Cecil Hubert Gostwyck, M.B., Ch.B., F.R.C.P.Edin., Dipl. Psych., M.P.C. Medical Superintendent, Moss Side State Institution, Maghull, near Liverpool.

1923. Gough, Isabel Falconer, M.B., Ch.B., L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., D.P.M., Shepherds, Cranbrook, Kent.

1928. Grace, Pierce, L.R.C.P.&S.I., Resident Medical Superintendent, District Mental Hospital, Portlaoighise (Maryborough), Leix.

1933. Graham, John James, M.B., Ch.B., D.P.M., Guildford Road, Lightwater, Surrey.

1914. Graham, Norman Bell, *M.C.*, B.A.R.U.I., M.B., B.Ch.Belf., D.P.M., Senior Assistant Medical Officer, Purdysburn Villa Colony, Belfast.

1918. Graham, Samuel John, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Resident Medical Superintendent, Purdysburn Villa Colony, Belfast.

1908. Graham, William Shepherd, M.B., B.Ch.R.U.I., Medical Superintendent, Somerset and Bath Mental Hospital, Cotford, near Taunton.

1921. Grant, Alastair Robertson, M.D., Ch.B.Aberd., Medical Superintendent, Lancashire County Mental Hospital, Whittingham, Preston.

1927. Grant, Florence Margaret (*née* Gamble), 306, St. James's Court, Buckingham Gate, S.W. 1.

1925. Grant, John King, M.B., Ch.B.Aberd., Assistant Physician, Royal Asylum, Montrose.

1930. Grant, Penuel Grant, M.B., Ch.B., D.P.H., Assistant Medical Officer, City Mental Hospital, Fishponds, Bristol.

1928. Graves (*née* Sykes), Kathleen Annie Harvey, M.D., B.S.Lond., M.B., Ch.B.Liverp., D.P.M., Rubery Hill, Birmingham.

1915. Graves, Thomas Chivers, B.Sc., M.D., B.S.Lond., F.R.C.S.Eng., L.R.C.P.Lond., M.R.C.V.S., Medical Superintendent, Rubery Hill and Hollymoor Mental Hospitals, Northfield, Birmingham.

1916. Gray, Cyril, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Senior Assistant Medical Officer, City Mental Hospital, Gosforth, Newcastle-upon-Tyne.

1928. Gray, James, M.D., ChB.Edin., Viewmount, Munlochy, Ross-shire.

1934. Greaves, Marion, M.R.C.S.Eng., L.R.C.P.Lond., D.P.M., Assistant Medical Officer, Cassel Hospital, Swaylands, Penshurst, Kent.

1909. Greene, Thomas Adrian, L.R.C.P.&S.Irel., *J.P.*, Medical Superintendent, District Mental Hospital, Carlow.

1936. Greener, Joan Ethne, M.B., Ch.B., D.P.H., Assistant Medical Officer, Bucks County Mental Hospital, Stone, near Aylesbury.

1922. Gregorson, Albert William, M.D., Ch.B., F.R.F.P.S.Glasg., Physician and Deputy Superintendent, North Middlesex Hospital, Edmonton, London, N. 18; 17, Forestdale, Southgate, N. 14.

1927. Grierson, Hugh Arrowsmith, *M.C.*, M.B., B.S.Lond., Medical Officer, H.M. Prison, Brixton, S.W. 2.

1926. Griffiths, Gwenvron Mary, M.D., M.R.C.P.Lond., 50, Wimpole Street, London, W. 1.

1934. Griffiths, Henry John, M.R.C.S.Eng., L.R.C.P.Lond., Assistant Medical Officer, Cefn Coed Hospital, Swansea.

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1901. Grills, Galbraith Hamilton, M.D., B.Ch.R.U.I., D.M.D., M.P.C., Medical Superintendent, Cheshire County Mental Hospital, Upton, Chester. (*Chairman, Northern and Midland Division*, 1936–37.)

1935. Guirdham, Arthur, B.A., D.M.Oxon., D.P.M., Medical Superintendent, Bailbrook House, Batheaston, Bath.

1922. Guppy, Francis Henry, *M.C.*, M.R.C.S., L.R.C.P., D.P.M., Deputy Medical Superintendent, Brighton Mental Hospital, Haywards Heath, Sussex.

1932. Hagedorn, Egmont Oscar, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Assistant Medical Officer, Stirling District Mental Hospital, Larbert.

1894. Halsted, Harold Cecil, M.D.Durh., L.S.A., M.R.C.S., L.R.C.P., Manor Road, Selsey, Sussex.

1935. Hall, Charlton Robert F., M.A., B.Ch.Camb., M.R.C.S., L.R.C.P., Medical Superintendent, Hellesdon Mental Hospital, Norwich.

1928. Hamilton, John Gerard, M.D., B.S., M.R.C.S., L.R.C.P., D.P.M., Senior Assistant Physician, Bethlem Royal Hospital, Monks Orchard, Eden Park, Beckenham, Kent.

1920. Hancock, Allen Coulter, *M.C.*, M.B., B.S., M.R.C.S., L.R.C.P.Lond., D.P.H., D.P.M., Medical Superintendent, Kent County Mental Hospital, Barming Heath, Maidstone.

1929. Handley, Richard, M.B., Ch.B.Manch., D.P.M., Medical Director, David Lewis Epileptic Colony, Warford, Alderley Edge, Cheshire.

1923. Hardcastle, Douglas Noel, M.R.C.S., L.R.C.P., D.P.M., 2, Lyndhurst Road, Hampstead, London, N.W. 3.

1920. Harding, Edward Palmer, L.R.C.P.&S.Irel., Deputy Medical Superintendent, East Riding Mental Hospital, Beverley.

1934. Hardwick, Sydney Walpole, M.D., B.Sc.Lond., M.R.C.P., D.P.M., Assistant Medical Officer, Horton Hospital, Epsom, Surrey.

1936. Harke, Wilfred Vaughan, M.R.C.S.Eng., L.R.C.P.Lond., D.P.M. (R.C.P.&S.), Honorary Medical Officer, Milford-on-Sea War Memorial Cottage Hospital; "Burwood", New Milton, Hants.

1932. Harkins, William, M.A., M.B., Ch.B.Aberd., Northcote, Strichen, Aberdeen.

1920. Harper, Raymond Sydney, M.R.C.S., L.R.C.P.Lond., 36, First Avenue, Hove, Sussex.

1904. Harper-Smith, George Hastie, M.A., M.D.Camb., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Brighton Mental Hospital; Galagate, Haywards Heath, Sussex.

1931. Harris, John Borrie, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Assistant Medical Officer, Worcestershire Mental Hospital, Barnsley Hall, Bromsgrove.

1924. Harris, John Stuart, M.D., Ch.B.Edin., M.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, West Park Hospital, Epsom.

1928. Harris, Noel Gordon, M.D., B.S., M.R.C.S., L.R.C.P.Lond., D.P.M., Finchley Cottage, East End Road, N. 2; 144, Harley Street, London, W. 1.

1898. Harris-Liston, Llewellyn, M.D.Brux., L.S.A., M.R.C.S., L.R.C.P.Lond., The Hall, Glan-y-don, Colwyn Bay, N. Wales.

1930. Harrison, Geoffrey J., L.R.C.P.&S.I., Lancs County Mental Hospital, Winwick, Warrington, Lancs.

1928. Harrowes, William McConnachie, M.D., Ch.B.Glasg., D.P.M., Medical Superintendent, New Saughton Hall, Polton, Midlothian.

1905. Hart, Bernard, M.D., F.R.C.P.Lond., M.R.C.S.Eng., Physician and Lect. on Ment. Dis., Univ. Coll. Hosp., 1, Harcourt House, Cavendish Square, London, W. 1.

1930. Haslam-Fox, Edward Arthur, M.B., Ch.B.Edin., D.P.M., Medical Superintendent, Cranage Hall, Holmes Chapel, Cheshire.

1892. Haslett, William John Handfield, M.R.C.S., L.R.C.P.Lond., M.P.C., *J.P.*, Resident Medical Superintendent, Halliford House, Sunbury-on-Thames.

1935. Haughie, Thomas McFadden, M.B., Ch.B., D.P.H., Assistant Resident Medical Officer, County Mental Hospital, Whittingham, Preston, Lancs.

1923. Hayes, Edmund Duncan Tranchell, B.A., M.D., B.Ch.Dubl., D.P.M., Medical Superintendent, County Mental Hospital, Berry Wood, Northampton.

1924. Hayes, Henry Douglas, M.D., Ch.B.Edin., D.P.M., Medical Superintendent, The Mental Hospital, Seacliff, Otago, New Zealand.

1920. Haynes, Horace Guy Lankester, M.R.C.S., L.R.C.P.Lond., Littleton Hall, Brentwood, Essex. (*Secretary, Parliamentary Committee*, 1929–36; *Chairman, S.E. Division*, 1934–36.)

1936. Haynes, William Noël Lankester, M.R.C.S.Eng., L.R.C.P.Lond., Lieutenant, R.A.M.C., Littleton Hall, Brentwood, Essex.

1935. Hayward, Sydney Thomas, M.B., B.S.Lond., L.R.C.P., M.R.C.S., Arthington, Barton Road, Torquay.

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1936. Hazell, Kenneth, M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Graylingwell Mental Hospital, Chichester, Sussex.
1927. Healey, Frederick Henry, B.Sc., M.D., Ch.B.Birm., D.P.M., Deputy Medical Superintendent, Somerset and Bath Mental Hospital, Cotford, Taunton.
1933. Hegarty, Denis, M.B., B.Ch., B.A.O.N.U.I., D.P.M., Assistant Medical Officer, Portrane Mental Hospital, Donabate, Co. Dublin.
1932. Hemphill, Robert Edward, M.B., B.Ch., B.A.O.Dubl., D.P.M., Assistant Medical Officer, City Mental Hospital, Bristol.
1920. Henderson, Cyril John, M.B.Durh., Medical Superintendent, The Royal Albert Institution, Lancaster.
1929. Henderson, David, M.D., Ch.B.Glasg., Assistant Medical Officer and Pathologist, Stafford County Mental Hospital, Cheddleton; The Hollies, Cheddleton, near Leek.
1916. Henderson, David Kennedy, M.D., Ch.B.Edin., F.R.F.P.S.Glasg., Physician Superintendent, Edinburgh Royal Mental Hospital, Morningside; Professor of Psychiatry, University of Edinburgh; Tipperlinn House, Edinburgh.
1923. Henderson, Norman Keane, B.A., LL.B.Camb., M.B., Ch.B.Edin., D.P.H., D.P.M., Medical Superintendent, Rauceby Mental Hospital, Sleaford, Lincs.
1930. Hennelly, Thomas John, M.D., B.Ch., B.A.O.N.U.I., D.P.M., Medical Superintendent, Cardiff City Mental Hospital, Whitchurch, Glamorgan.
1924. Hensman, Henry Saumarez, *O.B.E.*, L.M.&S.Madras, M.R.C.S., L.R.C.P.Lond., M.P.C., "Sargon House," No. 1, Atkinson Road, Vepery, Madras.
1924. Herbert, James Ewart, M.B., B.Ch.R.U.I., Senior Assistant Medical Officer, District Asylum, Omagh.
1925. Heron, John, M.B., Ch.B.Edin., D.P.M., British Dispensary, 35, Raffles Place, Singapore.
1929. Hewitt, Edward John Campbell, M.D., Ch.B.Edin., D.P.M., Deputy Medical Superintendent, Shenley Mental Hospital, Shenley, Herts.
1912. Higson, William Davies, M.B., Ch.B.Liverp., D.P.H., "The Elms", Parkhurst, Isle of Wight.
1934. Hill, Thomas Walter, M.D.Glasg., D.P.H.Camb., Deputy Medical Officer of Health and Certifying Officer under M.D. Acts, West Ham County Borough; Municipal Health Offices, Romford Road, London, E. 15.
1933. Hilliard (*née* Bunbury), Doris Elizabeth, M.B., B.S.Lond., M.R.C.P., D.P.M., 126, Harley Street, W. 1.
1933. Hilliard, Leslie Theodore, M.A., B.Ch., M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Tooting Bec Hospital, London, S.W. 17.
1931. Hills, Harold William, M.D., B.S.Lond., M.R.C.P., D.P.M.Camb., Honorary Physician and Anæsthetist, Stroud General Hospital; "Cotsmoor", Rodborough Common, Stroud, Glos.
1932. Hingston, Cicely Lamorna, M.R.C.S., L.R.C.P., *M.B.E.*(Mil.), Hon. Visiting Physician, Lady Chichester Hospital, Hove; 11, The Drive, Hove, Sussex.
1935. Holmes, Cedric, L.R.C.P., L.R.C.S.Edin., L.R.F.P.S.Glasg., L.D.S.Liverp., Assistant Medical Officer, County Mental Hospital, Chester.
1929. Holmes, Eric Gordon, M.A., M.B., B.Ch.Camb., Lect. in Biochem., Univ. Camb.; Biochemical Laboratory, Cambridge.
1925. Honan, Bernard Francis, L.R.C.P.&S.Irel., D.P.M., Medical Superintendent, District Mental Hospital, Cork.
1920. Hooper, Reginald Arthur, M.B., B.S.Durh., Medical Superintendent, City Mental Hospital, Fulford, York.
1929. Hopkins, Edwin Lancelot, *M.C.*, M.R.C.S., L.R.C.P.Lond., D.P.H., D.P.M., Medical Superintendent, County Mental Hospital, Mickleover, Derby.
1926. Hopwood, Joseph Stanley, M.B., B.S., M.R.C.S., L.R.C.P.Lond., Deputy Medical Superintendent, State Criminal Lunatic Asylum, Broadmoor.
1935. Horsley, John Stephen, M.R.C.S.Eng., L.R.C.P.Lond., Senior Assistant Medical Officer, Dorset Mental Hospital, Dorchester.
1926. Horton, Phyllis Mary, M.R.C.S., L.R.C.P.Lond., D.P.M., Michael House, 35, Lancaster Road, N.W. 3.
1918. Horton, Wilfred Winnall, M.D., C.M.Edin., Medical Superintendent, Wye House, Buxton.
1926. Hosie, William, M.B., Ch.B.Glasg., 2, Chester Road, Northwich, Cheshire.
1894. Hotchkis, Robert Dunmore, M.A.Glasg., M.D., B.S.Durh., M.R.C.S., L.R.C.P.Lond., M.P.C., Medical Superintendent, Renfrew District Mental Hospital, Dykebar, Paisley.
1934. Howie, James Erskine, M.D.Liverp., M.R.C.S., L.R.C.P., D.P.M., St. Fillans, Elton Head Road, St. Helen's, Lancs.
1904. Hughes, William Stanley, M.B., B.S., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Salop County Mental Hospital, Bicton Heath, Shrewsbury.

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1932. Hull, Eric Randal, M.B., B.Ch., B.A.O., D.P.M., Assistant Medical Officer, County Hospital, Lancaster.
1935. Hunnard, Frank Hwfa, M.R.C.S., L.R.C.P., 140, Croxted Road, W. Dulwich, S.E. 21.
1931. Hunter, Constance Primrose Helena, L.R.C.P.&S.Edin., D.P.H.Edin., Secretary, Scottish Association for Mental Welfare, 25, Palmerston Place, Edinburgh; St. Catherine's, Linlithgow.
1897. Hunter, David, M.A., M.B., B.Ch.Camb., L.S.A., Medical Superintendent, The Coppice, Nottingham. (*Secretary, S.E. Division*, 1910–1913).
1928. Hunter, James Dewar Hunter, M.B., Ch.B.N.Z., Dipl. Psych., Senior Assistant Medical Officer, Mental Hospital, Seacliff, Otago, New Zealand.
1935. Hurley, Theodore Egan, M.B., B.S., D.P.M., Assistant Medical Officer, Knowle Mental Hospital, Fareham, Hants.
1931. Hutton, Eustace, M.R.C.S., L.R.C.P., Deputy Medical Superintendent, North Wales Counties' Mental Hospital, Denbigh.
1911. Hutton, Isabel Emslie, M.D., Ch.B.Edin., Hon. Physician, British Hospital for Mental and Nervous Disorders; 6, Montagu Place, London, W. 1.

1926. Illingworth, Reginald Ernest, L.R.C.P.&S., L.D.S.Edin., D.Psy.Durh., Deputy Medical Superintendent, Northumberland County Mental Hospital, Cottingwood, Morpeth.
1915. Ingall, Frank Ernest, L.R.C.P.Lond., F.R.C.S.Eng., D.P.H., 36, Albert Road, Ashford, Kent.
1926. Ironside, Archibald Jennings, M.A., M.B., Ch.B.Aberd., Assistant Physician Superintendent, Fort Napier Institution, Pietermaritzburg, Natal, S. Africa.
1932. Irvine, Francis Philip, M.B., Ch.B.Liverp., Assistant Medical Officer, East Sussex Mental Hospital, Hellingly.
1906. Irwin, Peter Joseph, L.R.C.P.&S.Irel., Medical Superintendent, District Mental Hospital, Limerick.

1920. Jackson, John Luke, M.B., B.Ch.Belf., Medical Superintendent, Hants County Mental Hospital, Knowle, Fareham. (*Chairman, South-Western Division*, 1933–36.)
1936. Jackson, Winifred M., M.B., B.Ch., B.A.O.Belf., Assistant Medical Officer, Erdington House, Birmingham.
1927. Jacob, Frank Harwood, M.D., F.R.C.P.Lond., Hon. Physician, General Hospital; 32, Regent Street, Nottingham.
1927. Jacobson, Jack Nathan, M.R.C.S., L.R.C.P.Lond., D.P.M., D.M.R.E.Camb., Deputy Medical Superintendent, Fountain Hospital, London, S.W. 17.
1914. James, George William Blomfield, *M.C.*, M.D., B.S.Lond., L.S.A., D.P.M., Physician, Moorcroft House, Hillingdon, Uxbridge; 124, Harley Street, W. 1. Lect. on Ment. Dis., St. Mary's Hosp. (*Secretary, Parliamentary Committee*, 1926–29).
1935. James, Gwilym Treharne, B.Sc., M.B., B.Ch., Assistant Medical Officer, Dorset Mental Hospital, Herrison, Dorchester.
1922. Jarrett, Reginald Fitzroy, F.R.F.P.S.Glasg., L.M.S.S.A., Medical Superintendent, Leybourne Grange Colony, West Malling, Kent.
1908. Jeffrey, Geo. Rutherford, M.D., Ch.B.Glasg., F.R.C.P., F.R.S.Edin., M.P.C., Medical Superintendent, Bootham Park, York.
1925. Jenkins, John Alexander, M.B., Ch.B.Glasg., Medical Superintendent, Mental Hospital, Larbert.
1924. Jenkins, Reginald Edward, L.M.S.S.A., Willow Hayne, London Road, N. Cheam, Surrey.
1935. Johnson, James Stanley, M.B., B.S.Durh., D.P.M.Leeds, Assistant Medical Officer, Durham County Mental Hospital, Winterton, Stockton-on-Tees.
1893. Johnston, Gerald Herbert, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Brooke House, Upper Clapton, London, E. 5.
1928. Johnston, James McLauchlan, M.B., Ch.B.Edin., 28, Starbank Road, Edinburgh 5.
1936. Johnston, John Moore, M.B., B.Ch., B.A.O.Dub., Resident Medical Superintendent, The Mental Hospital, Omagh, co. Tyrone, Ireland.
1935. Jones, Maxwell Shaw, M.B., Ch.B., D.P.M., Assistant Physician, Royal Edinburgh Hospital for Mental and Nervous Disorders, Morningside, Edinburgh.
1936. Jones, Norman Howard, M.R.C.S., L.R.C.P.Lond., 90, Gloucester Terrace, Lancaster Gate, London, W. 2.

1934. Keane, Clarence Augustine, M.R.C.S.Eng., L.R.C.P.Lond., Assistant Medical Officer, Rubery Hill Hospital, Birmingham.
1927. Kearney, Joseph, M.B., B.Ch.N.U.I., D.P.M., Assistant Medical Officer, Mental Hospital, Letterkenny, Donegal.
1907. Keene, George Henry, M.D., B.Ch.Dubl., Medical Superintendent, Stewart Institution, Palmerston; 61, Cowper Road, Rathmines, Dublin.

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1909. Keith, William Brooks, *M.C.*, M.D., Ch.B.Aberd., M.P.C., Medical Superintendent, St. Audry's Hospital, Melton; Redwald House, Melton, Suffolk. (*Secretary, Parliamentary Committee*, 1921–26; *Chairman, S.E. Division*, 1936–7.)

1924. Kelly, Daniel Lane, L.R.C.P.&S.Irel., Inspector of Mental Hospitals, Local Government Department; 52, Grosvenor Road, Rathgar, Dublin.

1930. Kelly, John Vincent, M.B., B.Ch., D.P.M., Assistant Medical Officer, District Mental Hospital, Castlebar, Ireland.

1936. Kennedy, Alexander, M.D., B.S.Lond., M.R.C.P., Assistant Medical Officer, West Park Hospital, Epsom, Surrey; 124, Wickham Avenue, Cheam, Surrey.

1931. Kennedy, Robert Stewart, M.B., Ch.B., D.P.M., Deputy Medical Superintendent, Bracebridge Mental Hospital, Lincoln.

1902. Kerr, Neil Thomson, M.B., C.M.Edin., *J.P.*, Medical Superintendent, Lanark District Mental Hospital, Hartwood, N.B. (*Chairman, Scot. Div.*, 1930–31.)

1920. Key, Gordon James, M.B., Ch.B.Aberd., Medical Superintendent, Mental Hospital, Grahamstown, C.P., South Africa.

1923. el Kholy, Mohamed Kamel, M.R.C.S., L.R.C.P.Lond., D.P.M., Medico-Legal Department, Tanta, Egypt.

1920. Kimber, William Joseph Teil, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Herts County Mental Hospital, Hill End, St. Albans. (*Secretary, Educational Committee*, 1927–30.)

1903. King, Frank Raymond, B.A.Camb., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Peckham House, Peckham, London, S.E. 15.

1902. King-Turner, Arthur Charles, M.B., C.M.Edin., Medical Superintendent, The Retreat, Fairford, Gloucestershire.

1935. Kingston, Frank Edward, M.B., B.S., M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, West Riding Mental Hospital, Wakefield; 20, Bar Lane, Wakefield, Yorkshire.

1934. Kirkland, Hugh Thomson, M.A., M.B., Ch.B., D.P.M., Medical Officer, Erdington House; "Northolme", 192, Reservoir Road, Erdington, Birmingham.

1915. Kirwan, Richard R., M.B., B.Ch.R.U.I., Assistant Medical Officer, West Riding Mental Hospital, Menston, Leeds.

1933. Kirwan, William Joseph, M.B., B.Ch., B.A.O.N.U.I., D.P.M., Assistant Medical Officer, County Mental Hospital, Burntwood, Lichfield, Staffs.

1936. Kitching, Edwin Howard, M.B., Ch.B.Leeds, M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Cefn Coed Hospital, Swansea.

1919. Knight, Mary Reid, M.A., M.B., Ch.B.Glasg., Assistant Medical Officer, Paisley Mental Hospital, Riccartsbar.

1932. Krausz, Desider, M.D.Hungary, M.D.Bologna, Assistant Medical Officer, East Sussex Mental Hospital, Hellingly.

1914. Ladell, Robert George Macdonald, M.B., Ch.B.Vict., 615, Church Road, Yardley, Birmingham.

1923. Laing, John Kidd Collier, M.B., B.S.Melb., D.P.M., Medical Superintendent, Darenth Park, Dartford, Kent.

1935. Lalkaka, Kaikhushru Ardeshir J., M.B., B.S.Bombay, Hon. Psychiatrist to Bai Yamunabai Nair Hospital; "Nishat", Darabsha Road, off Nepean Sea Road, Bombay 6.

1925. Landers, John Joseph, M.B., B.Ch.N.U.I., D.P.H., Medical Officer, H.M. Prison, Princetown, Dartmoor, Devon.

1935. Langdon-Down, Mary, M.B., B.S.Lond., Assistant Medical Officer, Normansfield, Hampton Wick; Dixland, Teddington, Middlesex.

1896. Langdon-Down, Reginald L., M.A., M.B., B.Ch.Camb., Normansfield, Hampton Wick, Middlesex; Broom Close, Broom Road, Teddington.

1919. Langton, Peregrine Stephen Brackenbury, M.B., B.S., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Royal Earlswood Institution, Redhill, Surrey.

1925. Lascelles, William James, M.B., B.Ch.Belf., D.P.M., Assistant Medical Officer, Claybury Hospital, Woodford Bridge, Essex.

1935. Last, Samuel Leopold, L.R.C.P., L.R.C.S.Edin., L.R.F.P.&S.Glasg., M.D.Berlin, D.P.M., Assistant Medical Officer, The Mental Hospital, Berry Wood, Northampton.

1935. Laval, Evariste, M.B., C.M.Edin., Park End, Egham, Surrey.

1935. Laverty, Samuel John, B.A., M.B., Ch.B., B.A.O., L.M., D.P.M., Medical Superintendent, Harmston Hall Colony, Lincoln; Medical Adviser, Lincolnshire Joint Board for the Mentally Defective.

1929. Laws, John Joseph, M.R.C.S., L.R.C.P.Lond., D.P.M., Assistant Medical Officer, Horton Hospital, Epsom.

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1936. Leckie, James, B.Sc., M.B., Ch.B., Assistant Physician, Royal Mental Hospital, Aberdeen.
1915. Leech, Henry Brougham, B.A., M.D., B.Ch.Dubl., Deputy Medical Superintendent, Warwick County Mental Hospital, Hatton. (*Acting Registrar*, 1923–24.)
1909. Leech, John Frederick Wolseley, B.A., M.D., B.Ch.Dubl., D.P.M., Medical Superintendent, Wilts County Mental Hospital, Devizes.
1899. Leeper, Richard R., L.R.C.P., F.R.C.S.Irel., M.P.C., Medical Superintendent, St. Patrick's Hospital, Dublin. (*Secretary, Irish Division*, 1911–29, *and Chairman*, 1929–31; PRESIDENT, 1931–32.)
1906. Leggett, William, B.A., M.D., B.Ch.Dubl., Medical Officer, Smithston Mental Hospital, Greenock.
1927. Levinson, Reuben, M.B., Ch.B.Edin., D.P.H., D.P.M., Assistant Medical Officer, West Ham Mental Hospital, Goodmayes, Essex.
1933. Lewis, Aubrey Julian, M.D.Adelaide, M.R.C.P.Lond., Clinical Director, Maudsley Hospital, S.E. 5.
1930. Lewis, Edmund Oliver, M.A.Camb., D.Sc.Lond., M.R.C.S., L.R.C.P.Lond., Commissioner, Board of Control, Metropole Buildings, Northumberland Avenue, W.C. 2. (*Secretary, Mental Deficiency Committee since* 1935.)
1916. Lewis, Edward, F.R.F.P.S.Glasg., L.R.C.P.&S.Edin., Medical Superintendent, Hensol Castle Institution, Pontyclun, Glamorgan.
1924. Lewis, John Biddulph Strafford, M.A., M.D.Camb., M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, St. Bernard's Hospital, Southall, Middlesex.
1931. Liddell, John Kenneth Crawford, M.B., Ch.B.Edin., D.P.M., Senior Assistant Medical Officer, Barnwood House, Gloucester.
1920. Lilly, George Austen, *M.C.*, M.A., M.D.Camb., M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Cane Hill Hospital, Coulsdon, Surrey.
1927. Lindsay, Thomas, M.D., F.R.C.S.Edin., D.P.M., Medical Superintendent, Caterham Hospital (L.C.C.); The Pines, Caterham, Surrey.
1933. Linklater, George James Irvine, *O.B.E.*, M.D., M.R.C.P.E., D.P.H., D.T.M., Assistant M.O.H., Edinburgh; Westland House, Eskbank, Midlothian.
1908. Litteljohn, Edward Salterne, M.R.C.S., L.R.C.P.Lond., Medical Superintendent, The Manor, Epsom.
1921. Livesay, Arthur William Bligh, M.B., C.M., F.R.C.S.Edin., 497, Unthank Road, Norwich.
1935. Lloyd, John Ernest Seton, M.D., Ch.B.Liverp., M.R.C.P., D.P.M., Barrister-at-Law, Assistant Medical Officer, West Park Hospital, Epsom, Surrey.
1936. Lloyd, John Stanley, M.D., F.R.C.S.Edin., Hayes Park, Hayes, Middlesex.
1934. Lockwood, Madeline Rose, M.R.C.S., L.R.C.P., D.P.M., Resident Medical Superintendent, Fenstanton, Christchurch Road, Streatham Hill, S.W. 2.
1922. Logan, Frederick Colquhoun, M.B., Ch.B., F.R.F.P.S.Glasg., Medical Superintendent, County and City Mental Hospital, Gloucester; Lindi, Horton Road, Gloucester.
1932. Logan, William Robert, M.B., Ch.B., Assistant Superintendent, Mental Hospital, Singapore.
1924. Lornie, Peter, *O.B.E.*, M.D., Ch.B.Edin., Senior Assistant Medical Officer, Monmouth County Mental Hospital, Abergavenny.
1924. Lothian, Douglas B. M., M.B., Ch.B., F.R.C.P.Edin., D.P.M., Medical Superintendent, Down Mental Hospital, Downpatrick, N. Ireland.
1923. Lovell, Clement, *M.C.*, M.D., B.S.Lond., Pathologist, Bethlem Royal Hospital, Monk's Orchard, Beckenham, Kent; Beach Road, Emsworth, Hants.
1931. Lowenfeld, Margaret Frances Jane, M.R.C.S., L.R.C.P.Lond., Hon. Medical Director, The Children's Clinic for the Treatment and Study of Nervous and Delicate Children; 1, Weymouth House, Hallam Street, London, W. 1.
1906. Lowry, James Arthur, M.D., B.Ch.R.U.I., Medical Superintendent, Surrey County Mental Hospital, Brookwood.
1928. Lowson, William, M.B., Ch.B.St. Andr., Medical Officer, Moat House, Tamworth; 9, Colehill, Tamworth, Staffordshire.
1926. Lucas, Edmund Stanley Sayer, M.R.C.S., L.R.C.P.Lond., *I.M.S.*, c/o Grindlay & Co., Bombay, India.
1930. Lyons, Bernard, M.B., B.Ch., B.A.O.N.U.I., Medical Superintendent, District Mental Hospital, Enniscorthy, co. Wexford.
1936. Lyons, John Francis, L.R.C.P.&S.I., D.P.H., D.P.M., L.M.Ret., Medical Superintendent, Horfham Colony, Almondsbury, near Bristol.

1920. McAlister, William Malcolm, M.A., M.B., Ch.B., F.R.C.P.Edin., Dipl. Psych., Medical Superintendent, Bangour Village, West Lothian; Lect. on Psychiat., Univ. of Edin. (*Secretary, Scottish Division, since* 1935.)

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1906. Macarthur, John, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, County Mental Hospital, Bracebridge Heath, Lincs.

1923. Macaulay, Douglas Ian Otto, M.D., Ch.B.Edin., D.P.M., Medical Superintendent, Chiswick House, Pinner, Middlesex.

1936. McBroom, Robert P. J., M.B., Ch.B.Glas., Assistant Medical Officer, Argyll and Bute Asylum, Lochgilphead; Wedgwood, Cadzow Street, Motherwell.

1880. MacBryan, Henry Crawford, L.R.C.P.&S.Edin., Medical Superintendent, Kingsdown House, Box, Wilts.

1926. MacCallum, Alexander Grigor, M.B., Ch.B.Glasg., 8, Becmead Avenue, Streatham, London, S.W. 16.

1929. Maccallum, Archibald Montgomery, M.B., Ch.B.Glasg., Assistant Medical Officer, Ipswich Mental Hospital, Ipswich.

1929. MacCalman, Douglas Robert, M.D., Ch.B.Glasg., 128, Harley Street, London, W. 1.

1929. McCartan, William, M.D.Belf., M.R.C.P.Lond., D.P.H., D.P.M., Deputy Medical Superintendent, Banstead Hospital (L.C.C.), Sutton, Surrey.

1934. McCarthy, Cornelius J., M.B., B.Ch., B.A.O.N.U.I., B.Sc., D.P.H., D.P.M., Grangegorman Mental Hospital, co. Dublin.

1900. McClintock, John, L.R.C.P.&S.Edin., Resident Medical Superintendent, Grove House, Church Stretton, Salop.

1931. McConnell, Joyce, M.B., B.S.Lond., D.P.M., Assistant Medical Officer, Long Grove Hospital, Epsom.

1927. McCoull, George, M.D., B.S.Durh., L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Medical Superintendent, Prudhoe Hall Colony for Mental Defectives; Tyne View, Prudhoe-on-Tyne.

1936. McCowan, Alexander Knight, M.B., Ch.B., D.P.M., Assistant Medical Officer and Pathologist, Chartham Mental Hospital, Canterbury, Kent.

1920. McCowan, Peter Knight, M.D., Ch.B.Edin., F.R.C.P.Lond., D.P.M., Barrister-at-Law; Physician Superintendent, Crichton Royal, Dumfries. (*Secretary, Research and Clinical Committee, since* 1931.)

1926. McCowan, Mrs. P. K. (*née* Northcote), M.D., B.S., M.R.C.S., L.R.C.P.Lond., D.P.M., Crichton House, Crichton Royal, Dumfries.

1932. McCulley, William John, B.Sc., M.B., Ch.B., D.P.M., St. Andrew's Hospital, Thorpe, Norwich.

1936. McCulloch, Andrew Stenhouse, M.B., Ch.B.Edin., Dipl. Psych. Edin., Assistant Medical Officer, Lanark District Asylum, Hartwood.

1921. McCutcheon, Archibald Munn, M.B., Ch.B., F.R.F.P.S.Glasg., Medical Superintendent, Monyhull Colony, King's Heath, Birmingham.

1931. McDiarmid, Neil, M.B., Ch.B.Glasg., Medical Superintendent, Three Counties Mental Hospital, Arlesey, Beds.

1901. MacDonald, James Hogg, M.B., Ch.B., F.R.F.P.S.Glasg., Medical Superintendent, Glasgow District Asylum, Hawkhead, Cardonald, Glasgow; Lect. on Psychol. Med., Univ. of Glasgow. (*Chairman, Scottish Division*, 1932–33.)

1911. MacDonald, Ranald, *O.B.E.*, M.D., Ch.B.Edin., D.P.M., Medical Superintendent, Coton Hill Mental Hospital, Stafford.

1928. Macdonald, Robert William, M.B., Ch.B.Glasg., Medical Officer, Ministry of Pensions, Queen Alexandra Hospital, Cosham, Hampshire.

1905. MacDonald, William Fraser, M.B., Ch.B.Edin., M.P.C., Olive Lodge, Polworth Terrace, Edinburgh.

1931. MacDougall, Donald John, M.B., Ch.B., Major, *R.A.M.C.*, The British Military Hospital, Lucknow, India.

1928. McDougall, John, M.B., Ch.B.Glasg., Senior Assistant Physician, Crichton Royal, Dumfries.

1906. McDowall, Colin Francis Frederick, M.D., B.S.Durh., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Ticehurst House, Ticehurst, Sussex. (*Secretary, Library Committee, since* 1922.)

1895. Macfarlane, Neil M., *C.B.E.*, M.D., C.M.Aberd., Laurelhill, 10, Nonoti Avenue, Durban, Natal, South Africa.

1924. Macfarlane, Robert Melvin, M.D., Ch.B.Edin., D.P.H, D.P.M., Medical Superintendent, Jamnagar House, Staines, Middlesex. (*Secretary South-Eastern Division, since* 1935; *Secretary, Parliamentary Committee since* 1936).

1923. McGarvey, John, M.B., B.Ch.Belf., D.P.M., Medical Superintendent, Somerset and Bath Mental Hospital, Wells.

1928. MacGilp, Finlay Duncan, M.B., Ch.B.St. Andr., Deputy Medical Superintendent, Durham County Mental Hospital, Winterton, Ferry Hill, co. Durham.

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1934. McGlashan, Alice, M.B., Ch.B., D.P.H., Assistant Medical Officer, County Mental Hospital, Prestwich, Manchester.
1925. McGlashan, William Reid, M.A., M.B., Ch.B.Aberd., D.P.M., Physician to the Mental Health Services, Health Office, Lukis House, Grange, Guernsey.
1925. MacGown, Agnes Mildred, M.B., Ch.B.Edin., D.P.M., Assistant Medical Officer and Pathologist, Bangour Village, Uphall, Linlithgowshire.
1929. McGrath, Annie Margaret, B.A., M.B., M.R.C.S., L.R.C.P., D.P.M., Pathologist, Herts County Mental Hospital, Hill End, St. Albans.
1921. McGrath, Mathew Joseph, M.B., B.Ch.R.U.I., D.P.M., Deputy Medical Superintendent, West Riding Mental Hospital; Gortmore, Bar Lane, Stanley, near Wakefield.
1902. McGregor, John, M.B., Ch.B.Edin., Ardchoille, Llantrisant Road, Llandaff, Glamorgan.
1931. McIlroy, William Ernest, M.B., Ch.B., B.A.O., D.P.M., Stone House, St. Martins, Canterbury.
1924. McInnes, John, M.B., Ch.B.Glasg., D.P.M., Assistant Medical Officer, City Mental Hospital, Willerby, Hull.
1932. McInnes, Robert Gow, L.R.C.P.&S.Edin., M.R.C.P.Edin., Senior Assistant Physician, Royal Edinburgh Hospital; West House, Morningside, Edinburgh.
1921. McKail, Robert Buchanan Forbes, M.B., Ch.B.Glasg., D.P.M., Medical Superintendent, Brockhall Institution for Mental Defectives, Langho, near Blackburn.
1924. Mackay, George William John, M.B., Ch.B.Edin., D.P.M., Commissioner, Board of Control, Metropole Buildings, Northumberland Avenue, W.C. 2.
1929. MacKay, John, M.B., Ch.B.Glasg., D.P.M., County Mental Hospital, Whittingham, Preston, Lancs.
1914. Mackay, Magnus Ross, *M.C.*, M.B., Ch.B.Edin., Medical Superintendent, Newport Borough Mental Hospital, Caerleon, Mon.
1936. MacKeith, Stephen Alexander, M.R.C.S., L.R.C.P., D.P.M., Deputy Medical Superintendent, Warwick County Mental Hospital, Hatton.
1927. Mackenzie, Ivy, M.A., B.Sc., M.D., F.R.F.P.S.Glasg., Consulting Physician, Glasgow District Board of Control; 10, Woodside Terrace, Glasgow, C. 3.
1911. Mackenzie, John Cosserat, M.B., Ch.B.Edin., Assistant Medical Officer, Stafford Mental Hospital, Burntwood, Lichfield.
1926. Mackenzie, John Muir, M.B., Ch.B.Glasg., D.P.M., Deputy Medical Superintendent, Hollymoor Mental Hospital, Northfield, Birmingham.
1927. Mackenzie, Murdo, M.D., B.S., M.R.C.P.Lond., 41, Harley Street, London, W. 1.
1930. Mackenzie, Myra, M.B., Ch.B.Aberd., Medical Superintendent, The Lawn, Lincoln.
1934. Mackenzie, Norman Fraser, M.B., Ch.B.Edin., Assistant Medical Officer, Bracebridge Mental Hospital, Lincoln.
1903. Mackenzie, Theodore Charles, M.D., Ch.B., F.R.C.P.Edin., M.P.C., Druim, Inverness.
1935. McLaren, Robert Gordon Alexander, M.B., Ch.B.Aberd., B.Hy., D.P.H.Durh., Assistant Medical Officer, County Mental Hospital, Whittingham, Preston, Lancs.
1931. McLaughlin, Francis Leo, M.D., B.Ch.N.U.I., D.P.M., 146, Harley Street, London, W. 1.
1934. McLeman, John, M.B., Ch.B.Edin., c/o Wareing, Cropper, Son & Co., 61, Lord Street, Liverpool.
1927. Macleod, John, M.B., Ch.B.Edin., Assistant Medical Officer, Glasgow District Mental Hospital, Woodilee, Lenzie, N.B.
1921. Macleod, Neil, M.D.Edin., D.P.M., Medical Superintendent, The Retreat, York.
1925. McManus, Hugh Charles, M.B., Ch.B.Vict., D.P.M., 7, King Edward Avenue, Blackpool.
1930. McMenamin, Francis de Sales, *M.C.*, M.B., Ch.B.Edin., 38, Lower Baggot Street, Dublin.
1931. Macmillan, Donald MacPhail, M.B., Ch.B.Glasg., D.P.M., Medical Superintendent, Great Barr Park Colony, near Birmingham.
1929. Macmillan, Duncan, B.Sc., M.D., M.R.C.P.Edin., D.Psych., Deputy Medical Superintendent, Nottingham City Mental Hospital; Thorneywood Mount, Nottingham.
1925. MacNiven, Angus, M.B., Ch.B.Glasg., M.R.C.P.Edin., D.P.M., Physician Superintendent, Glasgow Royal Mental Hospital; 2, Whittingehame Gardens, Glasgow, W. 2. (*Secretary, Scottish Division*, 1934–35).
1910. MacPhail, Hector Duncan, *O.B.E.*, M.A., M.D., Ch.B.Edin., Medical Superintendent, City Mental Hospital, Gosforth, Newcastle-upon-Tyne; Lect. on Psychol. Med., Univ. of Durh.
1922. Macphail, Iain Ross, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Bootham Park, York.
1901. McRae, Douglas, M.D., C.M., F.R.C.P.Edin., *J.P.*, Physician-Superintendent, Glengall Hospital; Glengall House, Ayr, Scotland. (*Assistant Editor*, 1915–20, *and Co-Editor of Journal since* 1920; *Chairman, Scottish Division*, 1931–32; *Vice-Chairman, Educational Committee since* 1933; *President-Elect*, 1936–37.)

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1922. McWilliam, William, M.D., Ch.B., F.R.F.P.S.Glasg., D.P.M., Medical Superintendent, District Asylum, Inverness.
1925. Madgwick, John Reginald Alexander, M.D.Lond., M.R.C.S., L.R.C.P., D.P.M., Deputy Medical Superintendent, Long Grove Hospital, Epsom, Surrey.
1923. Madill, Joseph Thomas Herbert, B.A.R.U.I., M.B., B.Ch.Edin., F.R.F.P.S.Glasg., D.P.M., M.P.C., Medical Superintendent, Cumberland and Westmorland Mental Hospital, Carlisle.
1931. Magrath, Donald, M.B., Ch.B.Birm., M.R.C.S., L.R.C.P.Lond., D.P.H., D.P.M., 12, Cambridge Road, Ansdell, Lytham, Lancs.
1929. Mahony, Elizabeth Maud, M.B., Ch.B., B.A.O.N.U.I., Assistant Medical Officer, Mental Hospital, Monaghan, Ireland.
1928. Main, Dorothy Mary, M.B., Ch.B.Glasg., Assistant Medical Officer, City Mental Hospital, Willerby, Hull.
1931. Malloy, Joseph Holder, M.D.St. And., D.P.M., Deputy Medical Superintendent, Staffordshire County Mental Hospital, Cheddleton.
1908. Mapother, Edward, M.D., B.S.Lond., F.R.C.P.Lond., F.R.C.S.Eng., Medical Superintendent, The Maudsley Hospital, Denmark Hill, London, S.E. 5; Professor of Psychiatry, University of London, and Lect. in Psych. Med., King's Coll. Hosp.; 19, Queen Anne Street, London, W. 1.
1903. Marnan, John, B.A., M.B., B.Ch.Dubl., 2, Horton Road, Gloucester.
1929. Marsh, Rex Godfrey Blake, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Bromham House Colony, near Bedford.
1926. Martin, Alexander Reid, M.B., B.Ch.Belf., D.P.M., 24, Gramercy Park, New York City, U.S.A.
1922. Martin, Frederick Robertson, M.D., Ch.B.Glasg., D.P.M., 6, Grove Road, Sutton, Surrey.
1896. Martin, James Charles, L.R.C.S.&P.Irel., *J.P.*, 3, Sydenham Road, Ballsbridge, Dublin. (*Chairman, Irish Division*, 1936–37.)
1930. Martin, John James Black, M.A., M.D., B.Ch., M.R.C.P., D.P.M., Medical Superintendent, Fishponds Mental Hospital, Bristol.
1907. Martin, Mary Edith, L.S.A., L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., M.P.C., 11, The Drive, Hove, Sussex.
1914. Martin, Samuel Edgar, M.B., Ch.B.Edin., Barrister-at-Law, Medical Superintendent, The Old Manor, Salisbury. (*Secretary, South-Western Division, since* 1930.)
1921. Masefield, William Gordon, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Essex County Mental Hospital, Brentwood. (*Secretary, Educational Committee*, 1930–36; *Hon. General Secretary since* 1936.)
1911. Mathieson, James Moir, M.B., Ch.B.Aberd., Assistant Medical Officer, South Yorks Mental Hospital, Wadsley, Sheffield.
1934. Maudsley, Henry Fitzgerald, *M.C.*, M.D.Melb., M.R.C.P.Lond., D.P.M.Lond., Hon. Neurologist, Melbourne General Hospital; 8, Collins Street, Melbourne, Victoria, Australia.
1932. Maxwell, Robert Warnock, M.R.C.S., L.R.C.P., D.P.M., Surrey County Mental Hospital, Knaphill, Woking, Surrey.
1934. Maybin, Nora, M.B., B.Ch., B.A.O., D.P.H., Assistant Medical Officer, Rubery Hill Hospital, Birmingham.
1932. Menzies, Archibald, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Granby House, Youlgrave, Derbyshire.
1926. Menzies, Duncan, M.A., M.B., Ch.B.Aberd., D.P.M., Deputy Medical Superintendent, Plymouth Mental Hospital, Ivybridge, Devon.
1890. Menzies, William F., B.Sc., M.D., C.M.Edin., F.R.C.P.Lond., "Three Trees", Boreham Wood Road, Radlett, Herts. (*Vice-Chairman, Parliamentary Committee, since* 1932; PRESIDENT, 1920–21.)
1932. Methven, James Black, M.B., Ch.B.Edin., Assistant Medical Officer, Fife District Asylum, Cupar.
1910. Middlemiss, James Ernest, F.R.F.P.S.Glasg., M.R.C.S., L.R.C.P.Lond., M.P.C., "The Crest," Scott Hall Road, Chapel-Allerton, Leeds.
1928. Mill, Laura Margaret Dorothea, M.B., Ch.B.Glasg., 122, Craiglea Drive, Edinburgh, 10.
1929. Miller, Emanuel, M.A.Camb., M.R.C.S., L.R.C.P.Lond., D.P.M., Hon. Director, East London Child Guidance Clinic; 23, Park Crescent, London, W. 1.
1930. Miller, Frederick Richard Lanfear, M.R.C.S., L.R.C.P.Lond., D.T.M.&H., c/o J. F. C. Miller, Esq., 6, King's Bench Walk, Temple, E.C. 4.
1924. Miller, Robert Stewart, M.D., Ch.B.Glasg., Lebanon Hospital for Mental Diseases, P.O. Box 92, Asfuriyeh, near Beyrout, Syria.

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1931. Milmo, Dermod Hubert Francis, M.B., B.Ch.Dubl., D.P.H., Assistant Medical Officer, Caterham Hospital (L.C.C.), Caterham, Surrey.
1933. Minshull, Michael John Wilmott, B.M., B.Ch.Oxon., Assistant Medical Officer, Devon Mental Hospital, Exminster, Devon.
1923. Minski, Louis, M.D., B.S.Durh., M.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, St. Ebba's Hospital, Epsom, Surrey.
1922. Molony, Charles Bernard, M.B., Ch.B.N.U.I., D.P.M., Assistant Medical Officer, District Mental Hospital, Limerick.
1910. Monnington, Richard Caldicott, M.D., Ch.B.Edin., D.P.H., D.P.M., Neurologist, Ministry of Pensions; 33, New Street, Salisbury.
1915. Monrad-Krohn, G. H., B.A., M.D., B.S.Oslo, F.R.C.P.Lond., M.R.C.S.Eng., M.P.C., Prof. of Med., Royal Frederick University, Oslo; Rikshospitalet, Oslo, Norway.
1935. Montgomery, Neil McEachran, M.D., D.P.M., Senior Medical Officer, Storthes Hall Mental Hospital, Kirkburton, near Huddersfield.
1931. Moodie, William, M.D., F.R.C.P.Lond., D.P.M., Medical Director, London Child Guidance Clinic, Canonbury, N. 1; Associate Physician in Psych. Med., University College Hosp.; 128, Harley Street, London, W. 1.
1925. Moran, Patrick, M.B., B.Ch.N.U.I., D.P.M., Resident Medical Superintendent, Ardee Mental Hospital, Ardee, co. Louth.
1917. Morris, Bedlington Howel, M.B., B.S.Durh., Inspector-General of Hospitals, South Australia; "Tros-y-Parc", Pembroke Street, St. Peter's, Adelaide, South Australia.
1925. Morris, John Vincent, B.A., M.B., B.Ch.Dubl., Medical Superintendent, Little Plumstead Hall, near Norwich.
1896. Mould, Gilbert Edward, M.R.C.S., L.R.C.P.Lond., The Grange, Rotherham, Yorks.
1934. Moulson, Norman, M.D.Lond., D.P.M., Medical Superintendent, Cefn Coed Hospital, Swansea.
1914. Moyes, John Murray, M.B., Ch.B.Edin., D.P.M., Ardcaein, Shantallow, Londonderry.
1919. Mules, Annie Shortridge, M.R.C.S., L.R.C.P.Lond., Court Hall, Kenton, South Devon.
1907. Mules, Bertha Mary, M.D., B.S.Durh., Court Hall, Kenton, South Devon.
1929. Mullin, Bartholomew Joseph, *M.C.*, L.R.C.P.&S.Irel., D.P.H., D.P.M., Deputy Medical Superintendent, Wonford House Hospital, Exeter.
1929. Munro, Thomas Arthur, M.B., Ch.B., M.R.C.P.Edin., Dipl. Psych., Assistant Physician, Phipps Psychiatric Clinic, Baltimore, U.S.A.; 20, Merchiston Gardens, Edinburgh.
1925. Murdoch, James Wilson, M.B., Ch.B.Aberd., Medical Superintendent, Central Mental Hospital, Tanjong Rambutan, Perak, Federated Malay States.
1931. Murray, Helen Sara Euphemia, M.B., Ch.B.Edin., Assistant Medical Officer, Lancashire County Mental Hospital, Whittingham, Preston.
1932. Murray, John Raymund, M.D., B.S.Lond., M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Cane Hill Hospital, Coulsdon, Surrey.
1929. Muthiah, Asainayagam Richard, L.M.S.Singapore, Assistant Physician, Central Mental Hospital, Tanjong Rambutan, Perak, Federated Malay States.

1910. Neill, Alex. William, M.D., Ch.B.Edin., Physician-Superintendent, The Warneford Hospital, Oxford.
1920. Nicol, William Drew, M.B., B.S., M.R.C.P.Lond., D.P.M., Medical Superintendent, Horton Hospital, Epsom; Lect. on Psychiatry, London School of Medicine for Women.
1923. Nicole, J. Ernest, L.M.S.S.A., D.P.M., Senior Assistant Medical Officer, Lancashire County Mental Hospital, Winwick, Warrington.
1923. Nicoll, James, M.D., C.M.Edin., D.P.H., Medical Superintendent, Fountain Hospital, Tooting Grove, London, S.W. 17.
1920. Nix, Sidney, M.D., B.S.Durh., L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Deputy Medical Superintendent, Graylingwell Mental Hospital, Chichester.
1922. Noble, Ralph Athelstane, M.B., Ch.M.Syd., M.R.C.P.Lond., D.P.M., 86, Brook Street, Grosvenor Square, W. 1.
1909. Norman, Hubert James, M.B., Ch.B.Edin., D.P.H., Medical Superintendent, Camberwell House Mental Hospital, London, S.E. 5; Lect. on Ment. Dis., Westm. Hosp.; Northcotes, 79, West Hill, Sydenham, London, S.E. 26.
1932. Norris, Frank Edwin, M.R.C.S.Eng., L.R.C.P.Lond., D.P.M., Medical Officer, H.M. Prison; 164, Du Cane Road, London, W. 12.

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1930. O'Brien, Eveleen J., M.B., B.Ch., B.A.O.N.U.I., D.P.M., D.P.H., Assistant Medical Officer, Grangegorman Mental Hospital, co. Dublin.
1929. O'Connell, Daniel Joseph, M.D., B.Ch., D.P.M., Assistant Medical Officer, St. Andrew's Hospital, Northampton.
1934. O'Connor, Joseph, M.D., N.U.I., Assistant Medical Officer, Ardee Mental Hospital, Ardee, co. Louth.
1924. Odlum, Doris Maude, M.A.Oxon., B.A.Lond., M.R.C.S., L.R.C.P.Lond., D.P.H., 42, Harley Street, London, W. 1.
1918. Ogilvie, William Mitchell, M.B., C.M.Aberd., "Monymusk", 2, Riselaw Terrace, Edinburgh.
1911. Oliver, Norman Henry, M.R.C.S., L.R.C.P.Lond., Barrister-at-Law, Medical Superintendent, Ministry of Pensions Hospital; Northumberland House, Richmond, Surrey.
1936. Orchard, Ethel Adelaide, L.R.C.P.&S.Edin., c/o British Medical Association, Tavistock Square, London, W.C. 1.
1924. O'Reilly, James Joseph, M.B., B.Ch.Belf., D.P.M., Deputy Medical Superintendent, City Mental Hospital, Winson Green, Birmingham.
1930. O'Riordan, John Joseph, M.B., Ch.B., D.P.M., Assistant Medical Officer, North Riding Mental Hospital, York.
1902. Orr, David, M.D., C.M.Edin., M.P.C., 24, Grange Road, Edinburgh.
1910. Orr, James Henry Cubitt, M.D., Ch.B.Edin., Medical Superintendent, Midlothian and Peebles Asylum, Rosslyn Castle.
1933. Osburne, John Carr, M.B., B.Ch., B.A.O.N.U.I., Medical Superintendent, Lindville Private Mental Hospital, Cork.
1933. Osgood, Frederick Charles, M.B., Ch.B.St. And., Deputy Medical Superintendent, Royal Eastern Counties Institution, Colchester, Essex.
1936. O'Sullivan, Daniel Joseph, M.B., B.Ch., B.A.O.(N.U.I.), Assistant Medical Officer, Grangegorman Mental Hospital, Dublin.
1932. O'Sullivan, Edward N. M., B.A., M.B., B.Ch., B.A.O., D.P.M.N.U.I., Resident Medical Superintendent, District Mental Hospital, Killarney.
1935. Owen, David Rhyd, B.Sc., M.B., B.Ch.Wales, Medical Superintendent, Glamorgan County Mental Hospital, Bridgend; Angelton, Bridgend, Glam.

1928. Paddle, Kenneth Cecil Laurence, *M.C.*, M.R.C.S., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, Caterham Hospital (L.C.C.), Caterham, Surrey.
1930. Page, William Robert, B.A., M.B., Ch.M.Sydney, D.P.M.Lond., Hon. Psychiatrist, Sydney Hospital and St. Vincent's Hospital, Sydney; 221, Macquarie Street, Sydney, N.S.W., Australia.
1936. Palmer, Harold Anstruther, M.B., B.Ch., D.P.M., M.R.C.P., Senior Physician to Woodside Hospital, Woodside Avenue, N. 10.
1927. Pal, Sachindra Bhushan, B.A., L.M.S.Calc., Senior Assistant Physician, Central Mental Hospital, Tanjong Rambutan, Federated Malay States.
1933. Panton, John Barrie, L.R.C.P.Edin., Assistant Medical Officer, County Mental Hospital, Stafford.
1927. Parasuram, Govindarajpuram Rampattar, B.A., L.M.S.Madras, M.R.C.P.Edin., Deputy Medical Superintendent, Government Mental Hospital, Madras.
1932. Parfitt, David Neil, B.Sc., M.D., B.S.Lond., D.P.M., M.R.C.S., L.R.C.P., Medical Superintendent, Warwick County Mental Hospital, Hatton.
1931. Parker, William Patrick Hugh, L.R.C.P.&S.I., Assistant Medical Superintendent, Mental Hospital, Toowoomba, Queensland, Australia.
1920. Parkin, George Gray, M.D., Ch.B.Vict., Deputy Medical Superintendent, Cheshire County Mental Hospital, Parkside, Macclesfield.
1916. Patch, Charles James Lodge, *M.C.*, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Major *I.M.S.*, 39, Jail Road, Lahore, India.
1929. Paterson, Arthur Spencer, M.A.Oxon., M.D., Ch.B., F.R.C.P.Edin., M.R.C.P.Lond., 18, Harley St., W. 1.
1930. Paton, Thomas, M.D., Ch.B.Glasg., D.P.M., Deputy Medical Superintendent, St. Audry's Hospital, Melton, Suffolk.
1928. Patterson, Edward Cecil, M.B., B.Ch.Belf., Croom Lodge, Perry Hill, S.E. 6.
1931. Paul, Arthur Blackwell, M.A., M.B., B.Ch.Camb., Consulting Physician, Cheshire County Mental Hospital, Chester; Hon. Physician, Chester Royal Infirmary; Greyfriars House, Greyfriars, Chester.
1929. Pearce, John Dalziel Wyndham, M.A., M.D., Ch.B.Edin., M.R.C.P., D.P.M., Medico-Psychologist, London County Council; 13, Harley Street, London, W. 1.
1931. Pearce, May I., M.D., Ch.B.Birm., D.P.M., 17, Abbey Road, Harborne, Birmingham, 17.

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1920. Pearn, Oscar Phillips Napier, M.R.C.S., L.R.C.P., L.S.A., D.P.M., Deputy Medical Superintendent, Cane Hill Hospital, Coulsdon, Surrey.

1931. Pearson, Kate Young, M.B., Ch.B., Assistant Medical Officer, Rampton State Institution, Retford, Notts.

1913. Penny, Robert Augustus Greenwood, M.R.C.S., L.R.C.P.Lond., Deputy Medical Superintendent, Devon County Mental Hospital, Exminster.

1933. Penrose, Lionel Sharples, M.A., M.D.Camb., M.R.C.S., L.R.C.P., Research Medical Officer, Royal Eastern Counties Institution, Colchester; 47, Lexden Road, Colchester.

1927. Perera, Clement Osmund, M.R.C.S., L.R.C.P.Lond., D.P.M., Assistant Medical Superintendent, Mental Hospital, Angoda, Ceylon.

1928. Perk, David, M.D.Leeds, D.P.M., Assistant Medical Officer, West Riding Mental Hospital, Menston, near Leeds.

1929. Peters, Gordon Frank, M.B., B.S., M.R.C.S., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, Claybury Hospital, Woodford Bridge, Essex.

1911. Petrie, Alfred Alexander Webster, M.D., B.S., F.R.C.P.Lond., M.D., F.R.C.S.Edin., D.P.M., Medical Superintendent, Banstead Hospital (L.C.C.), Sutton, Surrey; Lect. on Ment. Dis., Charing Cross Hosp. and West Lond. Post-Grad. College. (*Vice-Chairman, Research and Clinical Committee, since* 1932; *Chairman, Educational Committee, since* 1933.)

1908. Phillips, John George Porter, M.D., B.S., F.R.C.P.Lond., M.P.C., Resident Physician-Superintendent, Bethlem Royal Hospital, Monk's Orchard, Beckenham, Kent; Physician and Lect. on Ment. Dis., St. Bart.'s Hosp.; 19, Cavendish Square, London, W. 1. (*Secretary, Educational Committee,* 1913–20.)

1906. Phillips, Nathaniel Richard, M.D.Brux., M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Monmouth County Mental Hospital, Abergavenny.

1905. Phillips, Norman Routh, M.D.Brux., M.R.C.S., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, St. Andrew's Hospital, Northampton.

1921. Phillips, Philip Gordon, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Bryn, The Avenue, Collingham, Leeds.

1924. Pickworth, Frederick Alfred, B.Sc., M.B., B.S., M.R.C.S., L.R.C.P.Lond., A.I.C.(exam.), Ph.C., Director, Joint Board of Research for Mental Diseases, City and University of Birmingham; Hollymoor Mental Hospital, Northfield, Birmingham.

1888. Pietersen, James F. G., M.R.C.S., L.R.C.P.Lond., Ashwood House, Kingswinford, near Dudley, Stafford.

1935. Pinkerton, William Maurice, M.B., B.Ch., D.P.H., Assistant Medical Officer, Wilts County Mental Hospital, Devizes.

1934. Pool, Arthur, M.B., Ch.B., M.R.C.P.Lond., D.P.M., Medical Superintendent, South Yorkshire Mental Hospital, Sheffield; Lecturer in Mental Disease, Sheffield Universty.

1913. Potts, William A., M.A.Camb., M.D.Edin.&Birm., M.R.C.S., L.R.C.P.Lond., Medical Officer, Birmingham Committee for the Care of the Feeble-Minded; 231, Hagley Road, Edgbaston, Birmingham.

1921. Poynder, Ernest George Thornton, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Plymouth Mental Hospital, Blackadon, Ivybridge, Devon.

1931. Prentice, David, M.B., Ch.B.Glasg., D.P.M., Assistant Medical Officer, Leicester and Rutland Mental Hospital, Narborough, near Leicester.

1936. Price, Annie Bapty, B.Sc., M.B., Ch.B., D.T.M.&H., Assistant Medical Officer, Chiswick House, Pinner, Middlesex.

1918. Prideaux, Joseph Francis Engledue, M.R.C.S., L.R.C.P.Lond., D.C.M.S.; Ministry of Pensions, 1, Sanctuary Buildings, Great Smith Street, London, S.W. 1.

1928. Pullar Strecker, Herbert A. J., M.D.Würzb., L.R.C.P.&S.Edin., Erlington, Kaimes Road, Edinburgh, 12.

1928. Quine, Margaret Annette, M.B., B.S., M.R.C.S., L.R.C.P.Lond., D.P.M., Assistant Medical Officer, Lancashire County Mental Hospital, Winwick, Warrington.

1930. Rae, James Burnett, M.B., Ch.B.Aberd., Hon. Physician in Charge, Department of Psychological Medicine, Croydon General Hospital; 93, Harley Street, London, W. 1.

1928. Raitt, William John, M.B., Ch.B.Aberd., Dipl. Psych., Senior Assistant Physician, Royal Mental Hospital, Aberdeen.

1894. Rambaut, Daniel F., M.A., M.D., B.Ch.Dubl., Medical Superintendent, St. Andrew's Hospital, Northampton; Priory Cottage, Northampton. (*Registrar since* 1924; *Chairman, South-Eastern Division,* 1933–34; PRESIDENT, 1934–35.)

1933. Rao, Bhaskara, M.R.C.S., L.R.C.P., Medical Officer, Government Hospital, Trivellore, Chingleput Dt., S. India.

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1936. Ratcliffe, Tom Arundel, B.A., M.B., B.Chir.Cantab., M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Nottingham County Mental Hospital; Birnam Lodge, Radcliffe-on-Trent, Nottingham.

1889. Raw, Nathan, *C.M.G.*, M.D., B.S., M.R.C.P.Lond., F.R.C.S., F.R.S.Edin., L.S.Sc.Durh., M.P.C., Lord Chancellor's Visitor; 22, Ashworth Road, London, W. 9. (*Chairman, Parliamentary Committee since* 1926; PRESIDENT, 1929–30.)

1913. Read, Charles Stanford, M.D., M.R.C.S., L.R.C.P.Lond., 9, Corringham Road, Golders Green, N.W. 11; Lect. on Psychol. Med. Bethlem Royal Hospital.

1920. Read, Walter Woolfe, M.D.Brux., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Berks County Mental Hospital, Moulsford, Wallingford.

1930. Rees, John Rawlings, M.A., M.D.Camb., D.P.H., Medical Director, The Institute of Medical Psychology; 37, Wimpole Street, London, W. 1.

1927. Rees, Thomas Percy, B.Sc., M.D., B.Ch.Wales, M.R.C.P.Lond., M.R.C.S.Eng., D.P.M., Medical Superintendent, Croydon Mental Hospital, Upper Warlingham, Surrey.

1911. Reeve, Ernest Frederick, M.B., B.S., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Lancashire County Mental Hospital, Rainhill, near Liverpool; Lect. on Ment. Dis., Univ. of Liverp.

1931. Reid, Benjamin, M.D., Ch.B.Glasg., D.P.M., Medical Superintendent, East Sussex Mental Hospital, Hellingly.

1911. Reid, Daniel McKinley, M.D., Ch.B., F.R.F.P.S.Glasg., Medical Superintendent, City Mental Hospital, Exeter.

1910. Reid, William, M.A.St.And., M.B., Ch.B.Edin., Medical Superintendent, Stafford Mental Hospital, Burntwood, Lichfield.

1932. Reid, William Muirhead, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., D.P.H., Assistant Medical Officer, Kingseat Mental Hospital, Newmachar, Aberdeen.

1929. Rich, Gilbert J., A.B., A.M., Ph.D.Cornell, M.D.Chicago, D.N.B., Director, Milwaukee County Mental Hygiene Clinic, 2430, West Wisconsin Ave., Milwaukee, Wisconsin, U.S.A.

1922. Riches, Reginald George, M.R.C.S., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, St. Bernard's Hospital, Southall, Middlesex.

1920. Rickman, John, M.A., M.D., B.Ch.Camb., Physician, London Clinic of Psycho-Analysis; 11, Kent Terrace, London, N.W. 1.

1931. Rivers, William Gregory, M.B., Ch.B.Edin., Medical Superintendent, Cornwall Mental Hospital, Bodmin.

1931. Roachsmith, C. E., M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Napsbury Mental Hospital, St. Albans.

1911. Robarts, Henry Howard, M.D., Ch.B.Edin., D.P.H., Medical Officer, East Lothian Mental Hospital, Ennerdale, Haddington, Scotland.

1922. Robb, John Robert Beith, M.D.Durh., L.R.C.P.&S.Edin., F.R.F.P.S.Glasg., Deputy Medical Superintendent, Glasgow District Mental Hospital, Gartloch, Gartcosh, Scotland.

1921. Roberts, Edward Douglas Thomas, M.R.C.S., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, Herts County Mental Hospital, Hill End, St. Albans.

1936. Roberts, John Herbert Owen, M.D., B.S.Lond., M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, North Wales Counties Mental Hospital, Denbigh; Carn Ingli, Denbigh.

1903. Roberts, Norcliffe, *O.B.E.*, M.D., B.S.Durh., D.P.M., Medical Superintendent, West Park Hospital, Epsom.

1935. Roberts, Reginald F., M.B., Ch.B., Medical Officer, County Mental Hospital, Rainhill; St. Fillans, Elton Head Road, St. Helens, Lancs.

1927. Robertson, David, M.D., Ch.B.Glasg., Assistant Medical Officer, Scalebor Park, Burley-in-Wharfedale, Yorks.

1908. Robertson, George Dunlop, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Dipl. Psych., 38, Kelvingrove Street, Glasgow, C. 3.

1934. Robertson, Guy Scott, M.B., Ch.B., Deputy Medical Superintendent, Calderstones, Whalley, near Blackburn.

1929. Robertson, James Andrew, M.B., Ch.B.Edin., 144, George Street, Hull.

1920. Robinson, William, M.D., Ch.B.Leeds, D.P.M., Medical Superintendent, City of London Mental Hospital, Stone, Dartford.

1936. Robson, Mary M., M.B., B.Ch., B.A.O.Belf., D.P.H., Assistant Medical Officer, Mental Hospital, Antrim, Ireland.

1922. Rodger, Kenneth Mann, M.B., Ch.B.Glasg., D.P.M., Medical Superintendent, Mental Hospital, Bulawayo, S. Rhodesia.

1914. Rodger, Murdoch Mann, M.D., Ch.B.Glasg., Dechmont, Helouan, Egypt.

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1930. Rodger, Thomas Ferguson, B.Sc., M.B., Ch.B.Glasg., D.P.M., 30, Falkland Mansions, Glasgow, W. 2.
1908. Rodgers, Frederick Millar, *O.B.E.*, M.D., Ch.B.Vict., D.P.H., Medical Superintendent, Lancashire County Mental Hospital, Winwick, Warrington.
1934. Rohan, James Charles, M.B., B.Ch., B.A.O.N.U.I., D.P.M., Assistant Medical Officer, Berks Mental Hospital, Wallingford, Berks.
1895. Rolleston, Lancelot William, *C.B.E.*, M.B., B.S.Durh., M.R.C.S., L.R.C.P.Lond., Queen Anne's Mansions, St. James's Park, London, S.W. 1.
1922. Rollins, Ernest Edward, B.A., M.B., B.Ch.Dubl., Resident Medical Officer, Brooke House, Upper Clapton, London, E. 5.
1931. Roper, William Francis, M.B., B.S.Lond., M.R.C.S., L.R.C.P., 26, Lyford Road, London, S.W. 18.
1934. Rose, Douglas John, M.R.C.S.Eng., L.R.C.P.Lond., First Assistant Medical Officer, Brockhall Certified Institution for Mental Defectives, Langho, near Blackburn, Lancs.
1935. Rose, Louis, M.R.C.S., L.R.C.P., Superintendent, Atkinson Morley Hospital, Copse Hill; c/o St. George's Hospital, London, S.W. 1.
1935. Ross, Charles MacDonald, M.B., Ch.B.Edin., Assistant Physician, Craig House, Morningside Drive, Edinburgh.
1888. Ross, Chisholm, M.D.Syd., M.B., C.M.Edin., 225, Macquarie Street, Sydney, New South Wales.
1910. Ross, Donald, M.B., Ch.B., M.R.C.P.Edin., M.P.C., *J.P.*, Medical Superintendent, Argyll and Bute Asylum; Tigh-na-Linne, Lochgilphead, Argyll. (*Vice-Chairman, Educational Committee*, 1927–33; *Chairman, Scottish Division*, 1936–37.)
1899. Rotherham, Arthur, M.A., M.B., B.Ch.Camb., Lord Chancellor's Visitor; "Hazards", Enton, Godalming, Surrey.
1922. Roy, John Allen Chisholm, M.B., Ch.B.Vict., Medical Superintendent, Royal Hospital, Cheadle, Cheshire.
1924. Rudolf, Gerald de Montjoie, M.R.C.P.Lond., D.P.H., D.P.M., 1, Victoria Square, Clifton, Bristol 8.
1929. Russell, David, M.D., Ch.B.Glasg., D.P.M., 24, Kempnough Hall Road, Worsley, near Manchester.
1923. Russell, John, M.B., Ch.B.Glasg., D.P.M., Assistant Medical Officer, West Riding Mental Hospital, Menston, Leeds.
1912. Russell, John Ivison, M.B., Ch.B., F.R.F.P.S.Glasg., D.P.M., M.P.C., Medical Superintendent, North Riding Mental Hospital, Clifton, York. (*Secretary, N. and M. Division, since* 1929.)
1915 Russell, William, *M.C.*, M.D., Ch.B.Edin., Dip. Psych., D.T.M., Commissioner for Mental Hygiene, Union Buildings, Pretoria, South Africa.
1912. Rutherford, Cecil, B.A., M.B., B.Ch.Dubl., Assistant Medical Officer, Holloway Sanatorium, Virginia Water, Surrey.
1907. Rutherford, Henry Richard Charles, F.R.C.S., L.R.C.P.Irel., D.P.H., Medical Superintendent, Farnham House, Finglas, co. Dublin.
1896. Rutherford, James Mair, M.B., C.M., F.R.C.P.Edin., M.P.C., Brislington House, Bristol.

1908. Samuels, William Frederick, L.M., L.Ch.Dubl., St. Dymphna's, Robyn's Way, Sevenoaks, Kent.
1932. Sanders, Marjorie Elizabeth Frances, M.B., Ch.B.Edin., D.P.M., Assistant Medical Officer, Long Grove Hospital, Epsom.
1935. Sands, Dalton Eric, M.R.C.P., L.R.C.S.Edin., L.R.F.P.S.Glasg., D.P.M., Assistant Medical Officer, Claybury Hospital, Woodford Bridge, Essex.
1923. Sang, Janet Adeline Agnes, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., Long Acre, Station Road, Scholes, Leeds.
1932. Schneider, Frank Edwin Ernest, M.D.Lond., M.R.C.S., L.R.C.P., D.P.M., Medical Superintendent, Rampton State Institution for Mental Defectives, Retford, Notts.
1928. Scholberg, Harold Alfred, M.B., M.R.C.S., L.R.C.P.Lond., D.P.H., Pathologist, Cardiff City Mental Hospital; 3, St. Andrew's Crescent, Cardiff.
1926. Scoresby-Jackson, Margaret, M.D., B.S.Durh., c/o Union Bank of Scotland, Ltd., 62, Cornhill, E.C. 3.
1925. Scott, Francis Leonard, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, City of Canterbury Mental Hospital, Canterbury, Kent.
1930. Scott, James, M.B., B.Ch., L.M., D.P.M., 8, Loraine Road, Holloway, N. 7.
1935. Scott, William Clifford Munroe, B.Sc., M.B., L.C.P.S.Ont., L.M.S.S.A., D.P.M., Assistant Medical Officer, Cassel Hospital, Swaylands, Penhurst, Kent.

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1911. Scroope, Gervace Wm. Mavy, M.B., B.Ch.Dubl., Resident Medical Superintendent, Central Criminal Asylum, Dundrum, co. Dublin.
1925. Selkirk, Elizabeth Thompson, M.B., Ch.B.Edin., Deputy Medical Superintendent, Hollymoor Mental Hospital, Northfield, Birmingham.
1929. Selling, Lowell Sinn, B.A.Mich., M.A.Columbia, D.N.B., Sc.M.New York, M.D.Bellevue, Ph.D., Psychopathic Clinic, Recorder's Court, City of Detroit; 16196, Cherrylawn Avenue, Detroit, Mich., U.S.A.
1912. Sergeant, John Noel, M.B., B.S., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Newlands House, Tooting Bec Common, London, S.W. 17. (*Secretary, South-Eastern Division*, 1913–35.)
1925. Shand, George Ernest, M.D., Ch.B.Aberd., D.P.H., Villa Belga, Westbourne Avenue, Bagatelle, Jersey.
1930. Sharpe, John Smith, M.B., Ch.B.Glasg., Assistant Medical Officer (Biochemist), County Mental Hospital, Stafford.
1901. Shaw, Benjamin Henry, M.D., B.Ch.R.U.I., Medical Superintendent, County Mental Hospital, Stafford. (*Chairman, N. and M. Division*, 1932–33; *Secretary, Research and Clinical Committee*, 1927–31.)
1905. Shaw, Charles John, M.D., Ch.B., M.R.C.P.Edin., *J.P.*, Medical Superintendent, Royal Asylum, Montrose. (*Chairman, Scottish Division*, 1933–34.)
1936. Shaw, David, M.D.Lond., M.R.C.P., Assistant Medical Officer, Claybury Hospital, Woodford Bridge, Essex.
1909. Shaw, William Samuel Jagoe, M.D.Belf., M.B., B.Ch.R.U.I., Lt.-Col. *I.M.S.* (ret.), Calcote House, Sandicotes Road, Parkstone, Dorset.
1920. Shearer, Christina Hamilton, M.B., Ch.B.Glasg., Senior Medical Officer, Cassel Hospital, Swaylands, Penshurst, Kent.
1928. Shepherd, Andrew, M.B., Ch.B.Glasg., D.P.M., Deputy Medical Superintendent, Worcester County Mental Hospital, Barnsley Hall, Bromsgrove.
1923. Shepherd, Charles Ernest Alan, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Kent County Mental Hospital, Chartham Down, near Canterbury.
1927. Shera, Arthur Geoffrey, M.A., M.D., B.Ch.Camb., M.R.C.S., L.R.C.P.Lond., Pathologist, Eastbourne Hospitals and East Sussex County Mental Hospital, Hellingly; 10, Upperton Gardens, Eastbourne.
1928. Sheridan, Alfred, L.R.C.P.&S.Irel., Resident Medical Superintendent, Mayo County Mental Hospital, Castlebar, Ireland.
1914. Sherlock, Edward Birchall, B.Sc., M.D.Lond., D.P.H., Barrister-at-Law, 20, Beresford Avenue, Twickenham, Middlesex.
1923. Shore, George William, *O.B.E.*, M.D.Lond., D.P.H. D.P.M., Medical Superintendent, Middlesex County Mental Hospital, Shenley, Herts.
1922. Shortt, Jane Elder, M.B., Ch.B.Glasg., Valleyfields, Alingdon, Lanarkshire.
1928. Silverston, Joseph Denzil, M.B., B.S.Durh., Deputy Medical Superintendent, County Mental Hospital, Lancaster.
1928. Sinclair, Arthur Crawford, M.D.Belf., D.P.M., D.P.H., D.M.R.E., Deputy Medical Superintendent, County Mental Hospital, Dorchester.
1921. Skene, Leslie Henderson, *M.C.*, M.B., Ch.B.Edin., Dipl. Psych., Medical Superintendent, Mental Hospital, Union Mills, Isle of Man.
1935. Skinner, Edward Fretson, M.A., M.B., B.Ch.Cantab., F.R.C.P., Physician, Sheffield Royal Hospital; 342, Glossop Road, Sheffield.
1925. Skottowe, James Stewart Ian, M.D.Glasg., D.P.M., Medical Superintendent, Bucks Mental Hospital, Stone, Aylesbury, Bucks.
1901. Slater, George Nathan Oscroft, M.D., M.R.C.S., L.R.C.P.Lond., D.P.M., Greystones, Long Itchington, near Rugby.
1934. Slight, David, M.B., Ch.B.Edin., F.R.C.P., D.P.M., Professor of Psychiatry, University Clinics Division of Chicago, The University of Chicago, U.S.A.
1933. Small, James Alexander, M.A., M.B., Resident Licensee, Heigham Hall, Norwich.
1928. Smith, Arthur Wallace Herbert, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Lunatic Asylum, St. Ann's, Port of Spain, Trinidad.
1934. Smith, Edward, M.B., B.S.Durh., Assistant Medical Officer, West Riding Mental Hospital, Menston, near Leeds.
1910. Smith, Gayton Warwick, M.D.Lond., B.S.Durh., M.R.C.S., L.R.C.P.Lond., D.P.H., Deputy Medical Superintendent, Springfield Mental Hospital, near Tooting, London, S.W. 17.
1905. Smith, George William, *O.B.E.*, M.B., Ch.B.Edin., Wyke House, Isleworth, Middlesex. (*Treasurer since* 1931.)
1933. Smith, Gilbert Mackay, B.A., M.B., B.Ch.Dubl., D.P.M., Assistant Medical Officer, City Mental Hospital, Mapperley Hill, Nottingham.

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1936. Smith, Henry St.George, M.A., M.D., B.Ch., B.A.O.(Dub. Univ.), Resident Medical Superintendent, The Stewart Institution, Palmerstown, co. Dublin.
1923. Smith, Herbert, M.R.C.S., L.R.C.P.Lond., Deputy Medical Superintendent, City Mental Hospital, Fishponds, Bristol.
1934. Smith, James Morrison, M.B., Ch.B., Deputy Medical Superintendent, York City Mental Hospital, Fulford, York.
1899. Smith, John Grimmond, M.D., C.M.Edin., " Strathmore ", Bowleaze Cove, Weymouth.
1931. Smith, Robert Sydney Steele, L.M.S.S.A., Assistant Medical Officer, Caterham Hospital (L.C.C.), Caterham, Surrey.
1913. Smith, Thomas Cyril, M.B., Ch.B.Edin., Deputy Medical Superintendent, County Mental Hospital, Barnwood, Gloucester.
1926. Snell, Harvie Kennard, M.D., B.S., M.R.C.S., L.R.C.P.Lond., D.P.H., Medical Officer, H.M. Prison, Brixton, London, S.W. 2.
1923. Somerville, George, M.D., Ch.B.Edin., D.P.M., Deputy Medical Superintendent, West Ham Mental Hospital, Goodmayes, Ilford.
1913. Somerville, Henry, B.Sc., M.R.C.S., L.R.C.P.Lond., F.C.S., Harrold, Bedford.
1906. Spark, Percy Charles, M.R.C.S., L.R.C.P.Lond., 15, South Croxted Road, Dulwich, London, S.E. 21.
1925. Speer, James Millar Craig, M.D., B.Ch.Belf., D.P.M., Deputy Medical Superintendent, Wilts County Mental Hospital, Devizes.
1929. Spence, David Sheridan, B.A., M.B., B.Ch., B.A.O.Dubl., Assistant Medical Officer, The Mental Hospital, Wells, Somerset.
1922. Spence, Thomas Reginald Carwardine, *M.C.*, M.B., Ch.B.Edin., Dipl. Psych., Medical Superintendent, Royal Scottish National Institution; Westerpark, Larbert, Stirlingshire.
1901. Starkey, William, M.B., B.Ch.R.U.I., Bryn-y-Neuadd, Llanfairfechan, N. Wales. (*Secretary, South-Western Division*, 1922–30.)
1936. Starr, Donald, M.B., Ch.B., Assistant Medical Officer, Chartham Mental Hospital, Canterbury, Kent.
1928. Statham, Hugh, M.B., B.C.Camb., M.R.C.S., L.R.C.P.Lond., Physician for Nervous Diseases, Royal Victoria and West Hants Hospital; 3, Elgin Road, Bournemouth.
1934. Steadman, Harry Hubert, M.B., B.S.Lond., M.R.C.S., L.R.C.P., D.P.M., Assistant Medical Officer, Claybury Hospital, Woodford Bridge, Essex.
1927. Steel, John P., M.D., Ch.B.Edin., Medical Superintendent, Smithdown Road Hospital, Liverpool.
1925. Steel, Samuel Maxwell, M.B., Ch.B.Glasg., Deputy Medical Superintendent, Monyhull Colony, Kings Heath, Birmingham.
1907. Steele, Patrick, M.D., Ch.B., F.R.C.P.Edin., Medical Superintendent, Roxburgh District Mental Hospital; The Hermitage, Melrose.
1929. Stenhouse, Jack Fingland Martin, M.B., Ch.B.Glasg., D.P.M., Assistant Medical Officer, Banstead Hospital (L.C.C.), Sutton, Surrey.
1929. Stephen, Adrian Leslie, B.A., M.B., B.S.Lond., Barrister-at-Law, Physician, London Clinic of Psycho-Analysis; 50, Gordon Square, London, W.C. 1.
1914. Stephens, Harold Freize, M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Birmingham Certified Institution, Coleshill Hall, Coleshill, near Birmingham.
1933. Stephenson, George Vaughan, M.B., B.Ch., D.P.M., Assistant Medical Officer, The Priory, Roehampton, London, S.W. 15.
1933. Stern, Edward Samuel, M.A., M.B., B.Ch.Cantab., M.R.C.P., D.P.M., Assistant Medical Officer, Napsbury Mental Hospital, near St. Albans, Herts.
1933. Stern, Ruby Olive, M.D.Lond., M.R.C.S., L.R.C.P., 23, New Cavendish Street, London, W. 1.
1909. Steward, Sidney John, *D.S.O.*, M.D., B.Ch.Camb., M.R.C.S., L.R.C.P.Lond., D.P.H., Dapdune, Guildford, Surrey.
1922. Stewart, Francis Hugh, M.A., D.Sc.St.And., M.D., Ch.B.Edin., D.P.M., Major *I.M.S.* (retired), Giza Memorial Laboratory, Cairo, Egypt.
1927. Stewart, Francis Melville, M.B., Ch.B.Edin., Deputy Medical Superintendent, County and City Mental Hospital, Littlemore, Oxford.
1931. Stewart, Ronald, M.B., Ch.B.Glasg., Deputy Medical Officer of Health for Mental Services, Corporation of Glasgow: 23, Montrose Street, Glasgow.
1887. Stewart, Rothsay Charles, L.S.A., M.R.C.S.Eng., " The Elms ", Oak End Waye, Gerrard's Cross, Bucks.
1914. Stewart, Roy MacKenzie, M.D., Ch.B., F.R.C.P.Edin., D.P.M., Medical Superintendent, Leavesden Hospital; Coles Farm, Leavesden, Watford, Herts.
1899. Stilwell, Reginald John, M.R.C.S., L.R.C.P.Lond., Moorcroft House, Hillingdon, Middlesex.

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1897. Stoddart, William Henry Butter, M.D., B.S., F.R.C.P.Lond., M.R.C.S.Eng., M.P.C., 57*a*, Wimpole Street, London, W. 1. (*Secretary, Educational Committee*, 1908–1912.)

1909. Stokes, Frederick Ernest, M.D., Ch.B.Glasg., D.P.H., " Glenmalure ", Portsmouth Road, Lee-on-the-Solent, Hants.

1885. Street, Charles Tidbury, M.R.C.S., L.R.C.P.Lond., Haydock Lodge, Newton-le-Willows, Lancs.

1929. Ström-Olsen, Rolf, M.D., B.Ch., B.Sc.Wales, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Runwell Hospital, near Wickford, Essex.

1909. Stuart, Frederick Joshua, *O.B.E.*, M.R.C.S., L.R.C.P.Lond., 55, Hillway, Highgate, London, N. 6.

1927. Stungo, Ellis, L.R.C.P.&S.Edin., L.R.F.P.S.Glasg., 16, Craven Park, Harlesden, London, N.W. 10.

1924. Sturrock, Alexander Corsar, M.A., M.D., C.M.Edin., M.R.C.P.Lond., Preston House, Eccles, Manchester.

1886. Suffern, Alex. Canning, *O.B.E.*, M.D., M.Ch.R.U.I., Glan-y-Mor, Hill Head, Fareham.

1921. Suffern, Canning, M.A., M.B., B.Ch.Camb., M.R.C.S., L.R.C.P.Lond., Glan-y-Mor, Hill Head, Fareham.

1922. Sullivan, Patrick Daniel, F.R.C.S., L.R.C.P.Irel., Medical Superintendent, Verville Asylum, Clontarf, co. Dublin ; 44, Harrington Street, Dublin.

1918. Sutherland, Francis, M.B., Ch.B.Edin., D.P.H., Deputy Commissioner, General Board of Control ; 25, Palmerston Place, Edinburgh.

1928. Sykes, Elizabeth Sarah Marples, M.B., Ch.B.Sheff., Assistant Medical Officer, South Yorks Mental Hospital, Wadsley, Sheffield.

1926. Talbot, Geoffrey, B.Sc., M.B., Ch.B.Manch., Assistant Medical Officer, Lancashire County Mental Hospital, Prestwich, Manchester.

1923. Tattersall, Stanley Roy, M.R.C.S., L.R.C.P.Lond., Deputy Medical Superintendent, Bucks County Mental Hospital, Stone, near Aylesbury, Bucks.

1931. Taylor, Arthur Leslie, M.D.Leeds, Ch.B., Hon. Pathologist, Bristol General Hospital ; Visiting Pathologist, Bristol Mental Hospital ; 83, Pembroke Road, Clifton, Bristol 8.

1910. Taylor, Arthur Loudoun, B.Sc., M.B., Ch.B., F.R.C.P.Edin., Senior Assistant Medical Officer, Stafford County Mental Hospital, Burntwood, Lichfield.

1935. Taylor, Fred Hayden, M.D., B.S., M.R.C.S., L.R.C.P., B.Hy., D.P.H., Medical Officer, H.M. Prison, Wormwood Scrubs ; 156, Du Cane Road, Shepherd's Bush, W. 12.

1924. Taylor, Frederic Cecil Marsh, M.R.C.S., L.R.C.P.Lond., D.P.M., Deputy Medical Superintendent, Kent County Mental Hospital, Chartham Downs, Canterbury.

1897. Taylor, Frederick Ryott Percival, M.D., B.S., M.R.C.S., L.R.C.P.Lond., 10, Carew Road, Eastbourne. (*Chairman, Educational Committee*, 1927–33 ; *Chairman, South-Eastern Division*, 1930–32.)

1935. Taylor, Moore, Major I.M.S., M.D., D.P.H., Medical Superintendent, Ranchi European Mental Hospital, P.O. Kanké, Bihar and Orissa, India.

1932. Taylor, Richard Hamilton, M.B., B.Ch., B.A.O., D.P.H.N.U.I., Pathologist, Grangegorman Mental Hospital, Dublin ; 119, Lower Baggot Street, Dublin.

1925. Taylor, Robert, L.R.C.P.&S.Irel., D.P.M., Assistant Medical Officer, St. Patrick's Hospital, Dublin.

1926. Tennent, Thomas, M.D., Ch.B.Glasg., D.P.H., D.P.M., Deputy Medical Superintendent, Maudsley Hospital, Denmark Hill, London, S.E. 5.

1921. Thomas, Cyril James, M.R.C.S., L.R.C.P.Lond., D.P.M., D.P.H., Medical Superintendent, West Riding Mental Hospital, Wakefield, Yorkshire.

1920. Thomas, Frederic Percival Selwyn, M.D., Ch.B.Vict., 4, Mill Ridge, Edgware, Middlesex.

1936. Thomas, John Clifford Sawle, L.R.C.P.Lond., M.R.C.S.Eng., Assistant Medical Officer, Bucks County Mental Hospital, Stone, near Aylesbury.

1908. Thomas, Joseph David, B.A., M.B., B.C.Camb., " Dan-y-Graig", Cheyne Road, Stoke Bishop, Bristol.

1911. Thomas, William Rees, M.D., B.S., F.R.C.P., M.R.C.S., D.P.M., M.P.C., Senior Commissioner, Board of Control ; 34, Chartfield Avenue, Putney Hill, London, S.W. 15. (*Vice-Chairman, Mental Deficiency Committee, since* 1935.)

1925. Thompson, Robert, M.B., B.Ch.Belf., D.P.M., Medical Superintendent, County Mental Hospital, Armagh, N. Ireland. (*Secretary, Irish Division since* 1929.)

1921. Thomson, Aidan Gordon Wemyss, M.B., Ch.B.Glasg., D.Psych., Deputy Commissioner, General Board of Control ; 25, Palmerston Place, Edinburgh.

1920. Thomson, William George, M.A., M.B., Ch.B.Aberd., D.P.H., D.P.M., Deputy Medical Superintendent, Royal Hospital, Cheadle, Cheshire.

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1927. Thorpe, Frederick Thomas, M.R.C.S., L.R.C.P.Lond., D.P.M., Assistant Medical Officer and Pathologist, South Yorkshire Mental Hospital, Wadsley, Sheffield.
1914. Tisdall, Charles Jerome, M.B., Ch.B.Edin., Medical Superintendent, Boughton Hall, Chester.
1935. Torrance, Lionel Stanley, M.B., Ch.B., D.P.M., L.M., District Medical Officer, and Public Vaccinator, Warwick County Council; "Dunedin", Kingsbury, near Tamworth, Staffs.
1896. Townsend, Arthur Allen Deykin, M.D., B.Ch.Birm., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Barnwood House, Gloucester.
1903. Tredgold, Alfred Frank, M.D.Durh., F.R.C.P.Lond., F.R.S.Edin., "St. Martin's," Guildford, Surrey; Lect. on Ment. Deficiency, London Univ.
1908. Tuach-Mackenzie, William, M.D., Ch.B.Aberd., Physician-Superintendent, Royal and District Asylums, Dundee; Lect. on Ment. Dis., Univ. of St. Andrew's; Westgreen House, Dundee.
1906. Turnbull, Peter Mortimer, *M.C.*, M.B., B.Ch.Aberd., D.P.M., Medical Superintendent, Tooting Bec Hospital, London, S.W. 17.
1909. Turnbull, Robert Cyril, M.D., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Essex County Mental Hospital, Severalls, Colchester.
1933. Turner, Cyril Edward Hedgman, M.R.C.S.Eng., L.R.C.P.Lond., Assistant Medical Officer, South Yorks Mental Hospital, Sheffield 6.
1906. Turner, Frank Douglas, M.B., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Royal Eastern Counties Institution, Colchester. (PRESIDENT, 1933–34; *Chairman, Mental Deficiency Committee, since* 1935.)
1922. Twomey, John Christopher, M.B., Ch.B.Liverp., D.P.H., Physician Superintendent, The Mental Hospital, Port Alfred, Cape Province, South Africa.
1934. Tyrrell, Thomas Anger Fereman, M.B., B.S.Lond., L.M.S.S.A., 9, Drayton Terrace, S. Kensington, S.W. 5.

1931. Valentine, James, M.B., Ch.B.Glasg., D.P.M., c/o Messrs. J. M. MacCormick & Neil, 121, Bath Street, Glasgow.
1922. Viehoff, Herman Crowther, M.R.C.S., L.R.C.P.Lond., 48, Moor Lane, Great Crosby, Lancs.
1894. Vincent, William James N., *C.B.E.*, M.D., B.S.Durh., M.R.C.S., L.R.C.P.Lond., 43, Westbourne Road, Sheffield, 10.

1923. Wadsworth, George Reginald, M.B., B.Ch.Belf., D.P.M., Assistant Medical Officer, Lancashire County Mental Hospital, Lancaster.
1928. Waldo, Henry Cecil, M.R.C.S., L.R.C.P.Lond., Barrister-at-Law, Medical Superintendent, Notts County Mental Hospital, Radcliffe-on-Trent, Notts.
1926. Walk, Alexander, M.D., B.S.Lond., D.P.M., Assistant Medical Officer, Cane Hill Hospital, Coulsdon. (*Assistant Editor*, 1928–31, *and Co-Editor of Journal since* 1931.)
1914. Walker, Robert Clive, M.D., Ch.B.Edin., Medical Superintendent, West Riding Mental Hospital, Menston, Leeds.
1908. Wallace, John Andrew Leslie, M.D., Ch.B.Edin., M.P.C., *J.P.*, Inspector-General of Mental Hospitals, New South Wales.
1912. Wallace, Vivian, L.R.C.P.&S.Irel., D.P.H., Ballinakill, Multyfarnham, co. Westmeath.
1932. Walsh, Fergus O'Connell, L.R.C.P.&S.Irel., Assistant Medical Officer, Portlaoighise (Maryborough) District Mental Hospital, Portlaoighise, co. Leix, Ireland.
1928. Walsh, Michael Anthony, L.R.C.P.&S.I., D.P.M., Assistant Medical Officer, Claybury Hospital, Woodford Bridge, Essex.
1889. Warnock, John, *C.M.G.*, B.Sc., M.D., C.M.Edin., M.R.C.S.Eng., The Limes, 181, London Road, Twickenham.
1922. Watson, Douglas Chalmers, M.D., F.R.C.P.Edin., Fenton Barns, Drem, East Lothian.
1924. Watson, John, *M.C.*, M.B., B.Ch.Edin., Resident Medical Superintendent, District Asylum, Londonderry.
1911. Webber, Leonard Mortis, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Surrey County Mental Hospital, Netherne, Coulsdon.
1929. Weber, Hilda Marion, B.Sc., M.D.Lond., D.P.M., Physician, The Institute of Medical Psychology; 23, New Cavendish Street, London, W. 1.
1922. Webster, William Leckie, M.B., Ch.B.Edin., M.P.C., Lt.-Col. *R.A.M.C.*, c/o Glyn, Mills & Co., Kirkland House, Whitehall, London, S.W. 1.
1936. Weir, Hugh, B.Sc., M.B., Ch.B., Assistant Physician, The House of Daviot, Pitcaple, Aberdeenshire.
1932. Weir, Thomas William Harold, M.D., B.Ch., B.A.O.Belf., D.P.H., D.P.M., Assistant Medical Officer, St. Audry's Hospital, Melton, Suffolk.

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1919. Westrup, Joseph Perceval, M.R.C.S., L.R.C.P.Lond., c/o Lloyds Bank, Ltd., Salisbury.

1911. White, Edward Barton Cartwright, M.R.C.S., L.R.C.P.Lond., St. Merryn, Braunton, N. Devon. (*Chairman, S.W. Division*, 1930–33.)

1921. Whitelaw, William, M.B., B.Ch.Glasg., 58, Hazelwood Road, Acocks Green, Birmingham.

1913. Wilkins, William Douglas, M.B., Ch.B.Vict., M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Stafford County Mental Hospital, Cheddleton, Leek.

1900. Wilkinson, Harry Bacon, M.R.C.S., L.R.C.P.Lond., "Dalestorth", Lynton Drive, Hillside, Southport, Lancs.

1929. Will, George Wishart, *O.B.E.*, M.B., Ch.B.N.Z., M.P.C., Major *R.A.M.C.*, "D" Block, Royal Victoria Hospital, Netley, Hants.

1925. Williams, Edward Lincoln, M.R.C.S., L.R.C.P.Lond, The Hall, Harrow Weald, Middlesex.

1922. Williamson, David Hardie, M.B., Ch.B.Edin., Assistant Medical Officer, Glasgow District Mental Hospital, Woodilee. Lenzie, N.B.

1923. Wilson, Alban, M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, Coldeast Colony, Sarisbury, Southampton.

1927. Wilson, Charles Herbert, M.B., B.Ch.Dubl., 6, Herbert Park, Donnybrook, Dublin.

1928. Wilson, Edward Alexander, M.D., Ch.B.Edin., Port Darwin, Falkland Islands.

1925. Wilson, Harriette Appleby, M.B., Ch.B.Leeds, D.P.M., Senior Assistant Medical Officer, West Riding Mental Hospital, Wakefield.

1930. Wilson, Henry Leonard, M.D., B.Ch.Camb., M.R.C.P., D.P.M., Physician, The Institute of Medical Psychology; 142, Harley Street, London, W. 1.

1923. Wilson, Isabel Grace Hood, M.D., Ch.B.Edin., D.P.M., Commissioner, Board of Control, Metropole Buildings, Northumberland Avenue, W.C. 2.

1920. Wilson, James Leitch, M.B., Ch.B.Edin., D.P.M., 10, New Cavendish Street, London, W. 1.

1899. Wolseley-Lewis, Herbert, M.D.Brux., F.R.C.S., L.R.C.P.Lond., "Dormers", Wimborne, Dorset. (*Secretary, Parliamentary Committee*, 1907–12; *Chairman*, 1912–21.)

1934. Wood, Alexander, M.B., Ch.B.Aberd., Deputy Medical Superintendent, County Mental Hospital, Newport, I. of Wight.

1912. Woods, James Cowan, B.A.R.U.I., M.D., B.S., M.R.C.S., L.R.C.P.Lond., 40, Harley Street, London, W. 1. Lect. on Ment. Dis., St. George's and London Hosps.

1885. Woods, John Francis, M.D.Durh., L.S.A., M.R.C.S.Eng., 7, Harley Street, London, W. 1.

1912. Wootton, John Charles, *M.C.*, M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Haydock Lodge, Newton-le-Willows, Lancs.

1922. Wootton, Leonard Henry, *M.C.*, B.Sc., M.B., B.S., M.R.C.S., L.R.C.P.Lond., D.P.M., Medical Superintendent, St. Ebba's Hospital, Epsom.

1900. Worth, Reginald, *O.B.E.*, M.B., B.S.Durh., M.R.C.S., L.R.C.P.Lond., Medical Superintendent, Springfield Mental Hospital, near Tooting, London, S.W. 17. Lect. on Ment. Dis., Westm. Hosp. (*Hon. General Secretary*, 1919–36; PRESIDENT, 1935–36.)

1917. Wright, Maurice Beresford, *O.B.E.*, M.D., C.M.Edin., Physician, The Institute of Medical Psychology; 86, Brook Street, London, W. 1.

1933. Wyatt, Walter, M.B., Ch.B.Edin., D.P.M.Lond., 10, The Avenue, Roundhay, Leeds 8.

1929. Wyllie, Andrew McNae, B.Sc., M.B., Ch.B., Clinical Pathologist, Crichton Royal Institution, Dumfries.

1928. Yates, Arthur Gurney, M.A., M.D.Edin., F.R.C.P.Lond., Physician, Sheffield Royal Infirmary; 53, Wilkinson Street, Sheffield.

1934. Yates, Irene, M.D., B.S.Lond., M.R.C.P.Lond., D.P.M., 501, White House, Albany Street, London, N.W. 1.

1921. Yellowlees, David, M.B., Ch.B.Glasg., 17, Lynedoch Crescent, Glasgow, C. 3.

1914. Yellowlees, Henry, *O.B.E.*, M.D., Ch.B., F.R.C.P.Edin., F.R.F.P.S.Glasg., F.R.C.P.Lond., D.P.M., Physician and Lect. on Psych. Med., St. Thomas's Hospital; 93A, Harley Street, London, W. 1.

1926. Young, Hubert Turner Penn, M.B., Ch.B.Edin., Medical Officer, H.M. Prison; 168, Du Cane Road, London, W. 12.

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OBITUARY.

Honorary Members.

1932. McDowall, Thomas W., M.D., L.R.C.S., "Burwood", Wadhurst, Sussex (*Ord. Mem. since* 1876; PRESIDENT, 1897–8.)

1935. Marr, Hamilton C., *C.B.*, M.D., C.M., F.R.F.P.S., M.P.C., Commissioner, General Board of Control for Scotland; 10, Succoth Avenue, Edinburgh. (*Ord. Mem.* 1896; *Secretary, Scottish Division*, 1907–10; PRESIDENT, 1927–28.)

1929. Pavlov, Ivan Petrovitch, M.D., Director of the Physiological Laboratories, Institute of Experimental Medicine and Academy of Science, Leningrad; Leningrad, v.o., 7 ligne, No. 2, U.S.S.R.

Corresponding Members.

1931. Jouchtchenko, Prof. A. J., Director, Ukrainian Government Institute of Clinical Psychiatry and Social Mental Hygiene, Kharkov, Ukraine, U.S.S.R., Konuchennaia 34.

Ordinary Members.

1912. Buchanan, William Murdoch, M.B., Ch.B.Glasg., Medical Superintendent, Kirklands Mental Hospital, Bothwell, Lanarkshire. (*Secretary, Scottish Division*, 1920–34.)

1920. Clark, Ronald Murray, M.B., C.M.Edin., 29, Irton Road, Southport.

1924. Craig, Alexander, M.B., Ch.B.Aberd., D.P.M., Assistant Physician, Royal Mental Hospital, Aberdeen.

1932. Flemyng, George Gilbert, M.D.Dubl. (retired), 7, Gambier Terrace, Liverpool.

1935. Limmex, Edith Gwendoline, M.R.C.S., L.R.C.P., D.O.M.S., D.P.M.Lond., Assistant Medical Officer, City Mental Hospital, Winson Green, Birmingham.

1877. Merson, John, M.A., M.D., C.M.Aberd., Willerby, Brayton Road, Selby.

1893. Mills, John, M.B., B.Ch.R.U.I., D.M.D., Medical Superintendent, District Mental Asylum, Ballinasloe, Ireland. (*Chairman, Irish Division*, 1935–36.)

1920. Smith, Maurice Hamblin, M.A.Camb., M.D.Durh., M.R.C.S., L.R.C.P.Lond., 190, Iffley Road, Oxford. (Lect. on Crim., Bethlem Royal Hosp.) (*Co-Editor of Journal since* 1931.)

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Part I.—Original Articles.

THE SEVENTEENTH MAUDSLEY LECTURE: THE BIOLOGY OF SOCIAL LIFE.

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I APPRECIATE very deeply the honour of being invited to deliver this the seventeenth Maudsley Memorial Lecture. I also feel the responsibility of adequately fulfilling a task so well carried out by my predecessors and so brilliantly initiated by Sir James Crichton-Browne. I never had the privilege of knowing Henry Maudsley, but Sir James's description of his fellow Yorkshireman as having "a dash of Wuthering Heights about him" illuminated him for me as by a flash of lightning. It has been said that his last book, *Organic to Human*, embodies his philosophy, which may be summed up in the principle of unity of the human organism and its continuity with the rest of Nature's processes. His *Pathology of Mind* also bears abundant testimony to his deep and life-long interest in the subject on which with some temerity I am venturing to address you. Whether I should have been rash enough to do so in his presence is another matter. I recall the fate of "a certain clergyman" whom Dr. Samuel Johnson blasted into silence by a single thunderbolt, and I fear that Dr. Henry Maudsley's keen and logical mind would pounce on some loose joints in my argument. So I crave your indulgence.

I have taken as my title "The Biology of Social Life". It is obvious that man, being a social animal, will be subject to the biological laws which govern life in general, and gregarious animals in particular. Yet how generally that simple fact is forgotten, although Wilfred Trotter has dealt, in his usual masterly style, with the instincts of the herd, and Morley Roberts with admirable skill has traced many biological and sociological parallels. I must acknowledge my indebtedness to them both by saying how stimulating their point of view has been to me.

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Looking at the admirable film made by the late Dr. Canti of the growth of bone under a microscope, one is struck by the ceaseless activity of the cells, busily engaged in building round the girders which are shooting out in all directions. Watching their apparently purposeful behaviour, it seems that when we say "my body", we are speaking rather as a king does when he says "my country" or "my subjects". Indeed, we know even less of the behaviour of our constituent cells then he does of the lives and deaths, the hopes and fears, the hates and loves of those over whom he rules.

It is a biological axiom that life started as a single cell, and still continues to do so. The most striking thing about a living cell is its incessant urge to assert itself as strongly as its environment will permit. This is the real struggle for existence. It is an extraordinarily interesting fact that if embryonic kidney-cells, for instance, are grown by themselves in a suitable medium they appear, according to some observers, to become positively malignant. Put some embryonic connective tissue into that medium and the kidney-cells promptly conform to type. They have to learn to adapt themselves to the needs of their neighbour. Cancer is a process of cell anarchy, the malignant cell brutally riding rough-shod over the others. Just so a man sufficiently released from the control of his environment becomes malignant, like many autocrats. As Mr. Baldwin said, "None of us is wise enough or good enough to be a dictator".

Maudsley wrote: "It is not easy for the individual to realize how much he owes to the restraints and supports of the social fabric in which he is an element, and which, like the atmosphere, always and insensibly surrounds him. There could not be a greater danger to the balance of any mind than to be exempt from the bonds and pressure of the surrounding social system." We see that contention of his illustrated in the United States to-day where, although one is particularly conscious of the pressure of herd opinion, there are outbreaks of gangsterdom, chiefly among imported aliens who have been set free from their own environment but have not assimilated themselves with the new, remaining Ishmaelites within it. It is one of the drawbacks of these vast new suburbs, mere dormitories, which radiate out like huge tentacles from London, destroying the countryside as they grow, that they offer so few opportunities for communal life and a social background.

The whole story of many-celled organisms is one of mutual adjustments between the different tissues, each trying to do its best for itself within the limits of those adjustments. The first stage in evolution was a number of single cells herding together for mutual support but each doing the same work. The next stage was one when groups of cells did different work. In evolution there are two parallel processes—increasing division of labour and increasing co-ordination between the different parts. The latter was achieved under the control of the central nervous system. A strong central government is needed to help to keep order, and no high degree of differentiation is possible

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in the animal body without the control of a centralized nervous system which has gradually acquired an increasing predominance. It is not too fanciful to compare the origin of the nervous system to a group of settlers on the coast, who gradually invade the interior, first singly and then in an organized army, as in the nervous system of vertebrates, which arises as a tube on the surface of the body. Once established, the invader assumes control over the indigenous inhabitants, fortifying itself as it goes, and maintaining its protectorate by a system of rapid communication throughout the invaded areas. The biological and sociological parallel is remarkably complete.

Just as the cells struggle to achieve the best they can within their environment, so the individual they form struggles either to do so, or to change its surroundings. The mud-fish gasping for breath on the mud-flats and struggling to reach the land was, no doubt, actuated by the need to escape from the competition of life in the sea towards the abundant food supply on the land. From that successful struggle all the land vertebrates and ultimately man himself arose. The power motive is therefore inherent in every cell in our body and is inherited from our remotest ancestors.

Now, in the evolution of the invertebrates a terrible dilemma arose—they are so constructed that their nervous systems cannot develop further without choking them. Two methods of escape from the dilemma were found : one, the development of the gregarious habit ; the other, the new pattern of nervous system which characterizes the vertebrate. The former method, in which each individual is absorbed into the community and is helpless apart from it, marks an advance which was fraught with great possibilities. For bees and ants this was comparatively easy, because of the very smallness of the brain of the individual and the limited number of reactions of which it is capable. Moreover the social habit in insects has imposed its demands, not only on the work, but on the structure of the individuals composing the group. It has sterilized large numbers, rendering them neuter and thus enormously simplifying the problem. Conflict and competition are greatly increased in a community where each individual aims at seeing himself immortalized in his offspring. Still more is this the case when one such community comes up against another similar one. The evolution of the vertebrate gave ample opportunities for the brain to expand. As the higher centres developed, the automatic actions of the lower centres were held in check while more skilled voluntary movements became possible. And as the highest level developed it exercised control over both voluntary and automatic movements, restraining emotional expression, but increasing skill through increased intelligence. Thus man, having laboriously acquired the power of speech, had to learn the still more subtle art of silence.

But long before man appeared, another " fault " in evolution had occurred. The first was the dilemma between nutrition and intelligence among the invertebrates ; the second was in the development of the mesozoic reptiles,

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in which the central nervous system was too rudimentary to control so huge a frame. Sir Herbert Samuel made some amusing play not long ago with the idea of such animals having an elaborate system of protection such as so often went with scanty brain power, and drew some political parallels consonant with his own views. The complete disappearance of these enormous animals (except perhaps from Loch Ness !) recalls how frequently the worship of the ideal of the colossal has heralded a downfall. There is something about them that inevitably suggests the prentice hand, just as there is about the first railway engine, the first motor car or the first " tank ". Anyhow, mere bigness was then exploited to the full and found wanting. Now it is important to note that on both these occasions a way of escape was found in gregariousness—the substitution of co-operation between smaller individuals, firstly for increased complexity of brain, and secondly for mere increased size of body.

The course of successful evolution has been to increase, not the size of the cell or of the individual, but of the unit. The unicellular became the multicellular ; isolated individuals became a community. For the mammal this was a much longer, more painful and more dangerous path than for the insect, just because its brain was capable of so many different reactions. This applies in an altogether special degree to man.

The first law of the herd is " Thou shalt not ". Just as the development of the higher nerve centres checks instinctive activities, so the development of communal life must restrict the freedom of the individual. Man has not found this easy. Philosophers may lament this, theologians may attribute it to original sin, but the biologist will remember that the cells of which he is composed did not find it easy to sink their individuality in that of the organism. A clever woman once said to me of her son, " He hasn't fused his ancestors yet ". It was profoundly true.

Yet evolution continues to demand that we shall fuse our ancestors, that we shall enlarge the unit. The family becomes the tribe, the tribe the small nation. The heptarchy becomes the monarchy, the nation an empire. And all the time the individuals within the unit are clamouring for self-expression, the smaller unit within the empire for self-determination. Sir Arthur Keith has called attention to two opposing tendencies in life—one the general demand for enlargement of the unit, the other the species-making impulse which attempts to segregate a particular type. The first in human life makes for internationalism, the second for nationalism.

The recent fierce fanatical outbreak of nationalism becomes comprehensible as a frightened response of the species-making segregating impulse against the resistless imperious demand of the ever-widening evolutionary process towards internationalism. More and more, nations become interdependent, the health and prosperity of one affecting the health and prosperity of all. Just as the revolt of the members against the belly became the defeat of all, so war has become a process in which all can lose but none gain. To win a

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war to-day is a greater disaster than it formerly was to lose one. Norman Angell pointed that out years ago; war would have been impossible if man were a rational animal. But to claim that man is a rational animal is to express a wish rather than to state a fact. Man is not a rational animal, though he may be in process of becoming so. But though reason is not the parent of many of his actions, he seeks reason for their god-parent, by trying to find reasonable explanations for what his emotions drive him to do. He prefers to ignore the savage ancestry of his unconscious mind. The outlook for increasing internationalism which we hoped for after the war is being replaced by a mentality resembling that of the Dobu islanders in Melanesia, whose slogan is, "If your yams flourish, mine will rot". In many respects popular ideas and ideals are undergoing a perilous repression. Here may I draw an analogy from Freud's doctrine concerning such repressions? If this species-making impulse is, as I think, repressing the evolutionary trend towards the expansion of the unit, one might expect that trend to take an abnormal course. The building up of vertical walls between nations is being countered by horizontal lines of cleavage, which threaten to divide Europe into Fascists and Communists. This, to my mind, represents the pathological form of internationalism resulting from repression of its normal expression.

Just as the nervous system must be developed to allow of sufficiently rapid co-operation between the various organs of the individual, so means of rapid communication are essential to co-ordination of the complex civilization of to-day. It has been truly said, "civilization *is* communication". One of the many factors in the downfall of the Roman Empire was the inadequacy of the methods of communication for the size it had attained. And so it suffered the fate of the mesozoic reptile. This difficulty does not obtain to-day. Means of communication could be excellent; so the forces which would oppose evolution offer artificial barriers to free communication—passports, visas, and the like. And all the time the development of aviation is making frontiers as obsolete as the octroi and the toll-gate. Indeed the great ease of communication by wireless is having some interesting effects. At first the leader could address personally all the members of a small tribe. The Greeks considered that the size of a city state was limited by the number of freemen who could assemble in a given place. Then as states increased in size various methods were evolved of appointing representatives of large groups to act for them. In democratic countries political power gradually passed into the hands of such representatives. But now the ruler can speak by wireless to a vast audience, which has facilitated a return to absolutism. We learned in the General Strike in 1926 what a powerful weapon wireless placed in the hands of government is, and dictators have not been slow to avail themselves of it.

In disease of the body, the higher levels which develop last suffer first, for they have not had time to acquire such a firm hold on the instincts of the organism as the lower. In the same way the body politic evolves its higher

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levels later than the individuals composing it. A committee is always more ready to perpetrate an injustice than its individual members would be. The laws of a nation may be fixed, but its ideas of international law are varied to suit its convenience. A contract between individuals may be indefeasible ; a treaty between nations is not. The contract may be enforced by superior authority ; the observance of a treaty, up to the present, has depended on force of arms. Yet this is no more logical than the settlement of a private quarrel by a duel. That it should be wrong for the individual to kill but right for the State to do so can only represent a transitional state in evolution. But in national affairs the human mind is still obsessed by the fallacy of force, though it has realized that in private affairs right does not necessarily lie with the better shot.

If we cannot adapt ourselves to the demands of evolution, the issue is not in doubt. This civilization will go, as others have gone. Flinders Petrie, in his fascinating little book on *The Revolutions of Civilization*, maintains that we are now in the eighth cycle of civilization. Each previous one has waxed and waned, and a dark age has intervened before the rise of the next. He regards as the real sign of progress throughout the ages the fact that the summers of the civilized periods are getting longer and the winters of the dark ages in between are getting shorter. He points out that the rise of each new civilization has originated " from a mixture of two different stocks. That effect of mixture cannot take place all at once. There are barriers of antipathy, barriers of creed, barriers of social standing, but every barrier of race fusion gives away in time when two races are in contact ". History gives many illustrations of the truth of this. Romeo and Juliet will always be impelled to break down the barriers between Montagus and Capulets. The classic civilization of the Greeks arose from the blending of a Northern with a Mediterranean stock. The Normans, a blending of Norsemen with Latins, were building cathedrals which are our admiration to-day at a time when their Scandinavian forbears were still building little wooden churches. Normandy, England and Sicily assumed new importance at their hands. With all the facts of the advantages of interbreeding at our disposal, how paltry and jejune is all this chatter about pure Nordic races which besets our ears to-day.

There are three stages in the evolution of human society ; to the first the name of " co-consciousness " has been applied, or, as Aldrich calls it, " a collective unconscious morality inherent in the laws of life ", so far as they can be appreciated. At this stage man is bound with a hypnotic completeness to every taboo. If he breaks a taboo he may even die, apparently from the sense of sin and isolation he experiences, as a bee dies when separated from the swarm. Rivers's studies of the Melanesians convinced him that they seemed to recognize instinctively, using that much-abused word in the strict sense, what the general feeling of the group was, and what definite line of action it should take. Such communities are stable precisely because they are not individualized. The

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avoidance of collisions between foot passengers in crowded pathways, thought reading and social tact, he regarded as vestiges among us to-day of that social common consciousness.

In the second stage individuals begin to emerge, and it is interesting to the medical profession to observe that it is the witch doctor, the magician, who is the first to do so. As Gerald Heard says, probably the proto-individual realizes that he is different from the herd before it becomes apparent to them. So he adopts a role which is impressive to the onlooker and suggestive to himself. But he is soon conscious not only that he is alone, and that he can never go back, but that there is enmity between him and the tribe, which spares him because it fears him. He evolves into the priest-king and, as Freud says, the elaborate taboos by which the lives of the priest-kings are made little better than a pestered imprisonment are the outcome of a profound subconscious jealousy, based on a feeling of fundamental difference in quality between the group and this particular individual. Frazer inquires, Why did it become customary in many parts of the world to put divine kings and other human gods to a violent death? It was because they feared that if they allowed the king to die of sickness or of old age, his divine spirit might share in the weakness of its bodily tabernacle, or perhaps perish altogether, thereby entailing the most serious danger to the whole body of the tribe. Whereas by putting him to death while he was yet in full vigour of body and mind, they could transmit his still uncorrupted powers to his successors. This is the real origin of the divinity that doth hedge a king, who was at first a sacrificial object—the central object of fertility rites on which the survival of the tribe depended. But as the evolution of human society proceeds, the individual learns to outwit the tribe, and to substitute others, or even an image or symbol for his own body, which was intended for sacrifice. Another man or an animal died in the king's stead. "The King is dead, long live the King." Such a change made for absolutism. The divine right went on without its distressing consequences.

This conception of kingship makes much more intelligible the ascription of divine powers and the final deification of the Cæsars, for this was merely a reversion to an earlier mode of thinking. It also explains their frequent assassination, which must often have seemed a religious duty to the assassin. Even to-day, in many parts of the world, assassination of the ruler is too often regarded as a political argument, and lunatics, who are admittedly prone to revert to primitive methods of thought, find a particular fascination in it.

The king at this stage in his attempts to outwit the tribe realizes that what its members want is physical plenty, and so he leads them out to conquest. A study of epic literature reveals that they are the record of a short violent, vivid period in the advance of human culture, an invariable stage, the heroic age. There is a transition from the cult of the totem animal to the heroic cult. "The glorious heroes are for the most part kings, but not in the old sense, bound to the soil, responsible for its fertility. Homage paid them is devotion

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for personal character . . . Another noticeable point is that in heroic poems scarcely anyone is safely and quietly at home." An heroic age is an almost invariable characteristic of the movements of people, and comes to an end with their re-settlement. We can therefore see how inevitable it is, even in modern times, that when a king who claims divine right leads his people out to battle unsuccessfully he should lose his crown, and often his head with it.

" The raiding stage over, the re-settled peoples will, to some extent, return to magic, as they will have gone back to crop-raising, and will once again be obsessed with fertility ; the generalship will break up ; a separation arises between the priestly and the kingly aspects. On the religious side, priesthood will re-establish itself, but as a profession. This priesthood will not have to pay its old price, neither will it be paid the old reverence " (Gerald Heard). The struggles between the kingly and priestly aspects of rulers are typified in the Middle Ages by the recurring conflicts between Emperor and Pope. They were seen in Egypt in the conflict between the " modernist " Aknaton and the priests of the temples. If the priest wins, he puts a boyish usurper on the throne, whom he can control. Such was Tutankhamen, who succeeded Aknaton. Seen from this angle what a fascinating story the history of the Old Testament becomes! Moses, followed by the victorious raiding general, Joshua ; the struggles between Samuel and Saul ; the successful claim of the priest to decide certain things against the king, as when Samuel hewed Agag in pieces before the Lord, but against the wish of Saul ; Saul, bewildered and outwitted by superior intelligence, reverts to fertility rites and consults the witch of Endor, and finally Samuel places the boyish usurper on the throne—David.

Originally only the king was regarded as having a future life, and therefore as the only one to possess a soul. But as individualism grew and spread, there was a similar demand from many humbler beings for immortality after death. Hence arose the mystery religions such as those of Osiris in Egypt, Orpheus in Greece, and Mithras in Persia, each of which purported to teach man how to attain to a future life. The spread of the Roman Empire imported these ideas into Rome itself, where, especially in the second century, they contested fiercely for supremacy with Christianity. And no impartial observer can deny that Christianity, in winning, absorbed some of the tenets of its rivals. St. Paul sometimes used the exact phraseology of Osiris and the Eleusinian mysteries, and hymnology, particularly Cowper's, is steeped in Mithraism. At the co-conscious stage the individual is satisfied with the continuance of the tribe, and fertility rites satisfy his religious aspirations. But at the stage of individualism he demands personal immortality. Fertility rites are regarded with grave disfavour and even disgust, and man's religious aspirations are fixed upon the next world. " In Israel, Josiah ends the fertility-religion with that sudden passionate revulsion against procreative rites which always

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attends such a termination, and which must show an abrupt amnesia of its own past."

And so individualism spreads and grows. In this second stage, as Aldrich says, " the group represses egotistic tendencies by forcibly imposing a conventional morality ". But as more and more individuals become self-conscious, the state becomes more unstable. It has been said that civilizations do not really decay, but burst from the tension produced by the rapid expansion of individualities within its borders. This is the present and urgent problem of civilization—to give scope for individual development, and yet for the individual to fit into his place as a part of a much larger whole. It is Aldrich's third stage—not yet reached by any society but recognized by an increasing number of persons—a stage in which the members of the group consciously co-operate for the common good, and not merely instinctively as in the social insects.

Heard emphasizes the point that whereas the first stage is accompanied by a fertility religion, while the second or individualistic stage is characterized by a religion in which personal salvation and personal immortality are the keynotes, neither of these is adequate for the third stage of conscious co-operation. Harold Nicolson considers that a struggle for world power creates conditions under which a new religion arises. And to-day, far removed as we are from a new world state, a new religion of the state as an object of worship is rapidly growing up. In Germany the old gods of Valhalla are being decorated with the armour of knighthood, just as during the war the wooden image of Hindenburg was erected as a totem figure into which nails were symbolically driven. The anti-religious attitude of Russia is upheld with a fanatical and intensely religious fervour, and all over Europe Fascism and Communism are fiercely competing religions.

Yet amid all change the human mind always desires to crystallize what should remain a living force, heedless of biology, which teaches that this is impossible. A living thing must always be changing—to crystallize is to kill the spirit and merely to preserve the letter. The human mind works with symbols, and as time goes on it is apt to take the symbol for the substance, instead of regarding it as an attempt, suited to the mentality of the age, to give expression to ideas, otherwise intangible. Nowhere is this more clearly seen than in a comparative study of religions. With the flux of time the old symbolic representation becomes unacceptable and often frankly repulsive to many individuals in subsequent generations. Yet to many, they represent the faith delivered once and for all, which it is sacrilege to question. Leonard Woolf puts it forcibly when he says, " The strangest and most important fact about communal psychology is that its content is largely the ideas, beliefs and aims of the dead . . . the law of the mortmain or the dead hand. . . . There can be no understanding of history, of politics, or of the effects of communal psychology which does not take into consideration the tremendous effect of this psychological dead hand, the dead mind. . . . At every

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particular moment it is the dead rather than the living who are making history, for politically, individuals think dead men's thoughts and pursue dead men's ideals . . . mere ghosts of beliefs, ideals from which time has sapped all substance and meaning. . . . A dogma is simply a belief which the living receive as a command from the dead".

But I would suggest that the real reason why this influence which the dead past continues to exert over the living man is so powerful, is that he carries within himself, still living, the genes of his dead ancestors. For, as Samuel Butler said in *Life and Habit*: "His past selves are living in him at this moment with the accumulated life of centuries. 'Do this, this, this, which we too have done and found profit in it', cry the souls of his forefathers within him. Faint are the far ones, coming and going as the sound of bells wafted to a high mountain; loud and clear are the near ones, urgent as an alarm of fire."

Thus is our psychological evolution limited and retarded. But unless we can overcome this difficulty sufficiently to adjust to the imperative demands of new conditions, the issue for civilization is scarcely in doubt. "Modify or disappear" is the inexorable sentence of evolution when the organism is confronted by a changing environment. Man feels the peril of change, and has sought to build breakwaters against the tide by means of written constitutions and printed creeds. The war was but the traditional seventh wave which smashed breakwaters that were already in peril. And in the deluge which accompanied and followed it man has lost confidence. Many things which he imagined were founded on rock he discovered were mainly built on sand.

A curious symptom of the prevailing disillusionment is the turning of the white races to the coloured ones for artistic inspiration: pictures of the South Sea Islanders, carved wooden images from Tahiti, jazz music, negro spirituals. The world resounds with dark laughter, as the white man uneasily shifts the burden on his shoulders. Some of the reasons for this disillusionment and loss of confidence are obvious; others are more deeply seated. Man's reaction to Nature varies with the control which he feels he has over it.

Recently man has become rather overawed by the universe in which he finds himself. He can hardly comprehend the vastness and emptiness of the interstellar spaces, or the minuteness of the electrons within the atom. Life trembles, as it were, in a narrow zone between intolerable heat and intensest cold; if it wavers on either side, it ceases to be. It can only exist in association with an atom which holds twelve electrons within its orbit. Staggered by such facts, he is too apt to forget that the most marvellous of all matter is the nerve-cell, and that, so far as we know, he possesses the most highly developed system of such cells, whereby he can perceive and interpret the phenomena by which he is surrounded. The astronomer in H. G. Wells's story realized that he was greater than the comet which was presently going to destroy the earth and him with it, because he knew what the comet was going to do, and the comet did not.

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But man has become much less confident of the control which reason can exert over his instincts. It required the convulsion of a great war abruptly to remind us that if we had subdued Nature externally, internally, in ourselves, she is as cruel and bloodthirsty as ever. Man has acquired a control over machines without acquiring anything like a corresponding control over himself. He does not even appear to be able to control satisfactorily something he has created for his own convenience—namely currency. He has a fatal aptitude for applying his discoveries to destructive ends. Aviation has been described recently as " the discovery that took the wrong turning ", and that is typical of much.

Evolution always offers a higher and a lower road : to a form which involves an expanding, more complex unit, or to one which degenerates to a lower level, though more consonant with the capacity of its components. The ascidian, reduced to a jelly-like lump clinging to a rock like a piece of sea-weed, still retains vestiges of its vertebrate origin, and its young start off in each generation as hopeful young vertebrates, until the fatal taint of degeneration strikes them. But that is to look at it from our point of view. The ascidian lazily clinging to that rock may be perfectly happy. This is what makes present-day conditions so disturbing. Whole nations which apparently feel unable to maintain the ideals that we regard as the higher ones actually seem to gain a new hope and a new faith by departing from them. The new level is more suited to their evolutionary development, and they are more comfortable in it. Depreciation of ideals, like depreciation of currency, seems to give them a new stability.

In this process no ideal has suffered so severely as that of liberty. Harold Nicolson inquires, " Is national independence so far more important than personal freedom ? That is a question ", he went on to say, " which the present generation are unable to answer ". It seems to me that a good many nations have already answered it in the affirmative. The segregating, species-making impulse has at any rate for the moment the upper hand. General Smuts recently made a powerful claim for liberty as the foundation of political health and happiness ; whereas in three-fourths of Europe liberty is now regarded as a curse, tyranny as the way of salvation. It may be that the old ideas of liberty were too negative ; it may well be that the population of large areas of the globe are not yet fit for it. We feel it about India, so we need not be surprised if some European countries feel it about themselves. It is certainly the fact that nineteenth century ideas of liberty were too much bound up with a policy of *laisser faire* which led to social injustice. It is inevitable that the State shall take an increasing part in planning the lives of its individual citizens. In the same way we are learning that in the body the central nervous system exercises some control, even over functions which were thought to be entirely autonomous. I reminded you at the outset that in evolution there are two parallel processes at work—increasing division of labour and increasing

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co-ordination. The evolution of large industries illustrates both of these. Indeed, the division of labour in a large factory has reached such a pitch that in many occupations craftsmanship is dead and the workman has become a robot. It is therefore significant and encouraging to find that a captain of industry, Sir Josiah Stamp, should have devoted his presidential address to the British Association to a consideration of the effects of industrial change upon the social organism—a subject of great importance to the welfare of the State. He pointed out that when the time-span of important change was considerably longer than that of the individual human life, we enjoyed the illusion of fixed conditions. Now the time-span is much shorter, but our attitude of mind still tends to regard change as the exceptional, and rest as the normal, rather than the converse. But these alternative attitudes make all the difference to the accommodating mechanisms we provide. In one case there will be well-developed tentacles, grappling irons, anchorages and all the apparatus of security. In the other, society will put on casters and roller bearings, cushions and all the aids to painless transition. Thus the written Constitution of the United States, devised for the "horse and buggy" days, acts as a serious drag on adaptations to entirely different conditions. A scientific study of the effects of scientific discovery itself on both capital and labour is urgently needed in order to render the change from obsolescent to new methods as smooth as possible. As Sir Josiah says, "Since man does not live by bread alone, if a ruthless industrial organization continually tears up the family from its roots, transferring it without choice to new surroundings, destroying the ties of kin, home and social life, of educational and recreational environment, it is far from ideal. Human labour can never be indefinitely fluid and transferable in a society that has a soul above consumption of mere commodities". He goes on to discuss the factors determining the optimum conditions of change, pointing out that a natural increase of population is both the best shock-absorber and safety valve that the community can possess, and we are faced with the problem that in all Western industrial countries the population will shortly become stationary, and then will noticeably decline. He is content to point out the effects of this, and does not indulge in the scolding which we usually have to listen to when the falling birth-rate comes under discussion. Looked at broadly, is it not probable that after the enormous increase of population in the nineteenth century there should be a lull? I do not know whether anthropologists are prepared to explain the outburst of fertility in north-eastern Europe and western Asia in the early centuries A.D. which swept aside all barriers, inundated the Roman Empire and dissolved it into fragments. One can only vaguely visualize the enormous pressure of population that initiated these huge waves of migration. But they did not continue indefinitely, and the population of England was neither great nor rapidly increasing in the days of Queen Elizabeth, which was certainly not an age of decadence. If our industrial system really depends for its success upon

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an increasing number of individuals to produce an increasing amount for an increasing population to consume, it is bound to crash sooner or later. Fortunately Sir Josiah leaves us a loophole by saying that the effects of a static population can be mitigated if the *per capita* income is increasing. Because as the parts of the globe inhabitable by the white races fill up, the population will inevitably become static. Whether the present lull is entirely due to contraceptive practices or whether there is also some fall in natural fertility it is impossible to say ; certainly medical men see plenty of childless parents who ardently desire offspring. Carr-Saunders, in his recent book, *World Population*, thinks it likely that the time may soon come when among white people no more children will be born than are desired before conception. One reviewer, horror-struck at such an idea, said this may mean a far more rapid crumbling away even than that in Great Britain at present, whereas for my part I should expect it to solve many of our psychological problems. Anyhow, if that is the natural trend, neither threats nor bribes will alter it ; indeed one might point out that if finance were all, the bribes are so paltry that they would only influence the unthinking type of person, who would welcome a shilling even if he knew he would have to pay a sovereign for it later on.

It is gratifying at any rate to be told that a higher standard of wealth may compensate for a falling population to some extent, for I believe that this higher standard and more settled conditions would lead to the production of more children to enjoy them, and thus with minor fluctuations the balance would be maintained. But how dangerous is prophecy! I am grateful to Mr. J. A. Spender for reminding us of a book entitled *National Life and Character*, by Charles Pearson, which was published in 1893. I remember being greatly impressed by it at the time. Looking ahead, Pearson saw the world becoming duller and quieter ; war would only be occasionally resorted to when arbitration had failed, and even then it was possible to hope that it would be conducted without intentional injury to non-combatants, and with the smallest possible damage to private property. There would be socialism of a limited, bureaucratic and unprogressive sort ; science had done its greatest work, and the future could have no great discoveries in store ; the general tendency would be towards the static and the somnolent, and so on. Those were the views of a learned and experienced man. Yet as Spender says, " If we wished to find a statement, exact in almost every detail of what has not happened in the subsequent forty years we could hardly do better than go to this book. . . . Greater and more sudden changes have come to pass than in any similar period of history. . . . To the Victorian Liberal who thought of liberty and progress as things established beyond possibility of challenge, the state of several great nations in Europe would have seemed like an eruption from the nether regions ". Nor is this change incompatible with what I previously said about the influence of the dead hand, for we are experiencing its regressive

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drag on an age furnished with more powerful weapons of destruction to assist in its atavistic savagery.

I hope I have made clear what seems to me the present dilemma. The first biological stage of a community is held together by taboos and fertility rites; individuality is at a discount. In the second, individualities develop, and the religious trend is towards individual salvation and personal immortality. The increasing tendency towards individualism becomes incompatible with the highly organized state which a complex civilization requires, for, as Maudsley said, the social organism of which the individual is an element only exists by some suppression of his purely self-regarding impulses. The evolutionary demand for an enlargement of the unit excites a defensive reaction towards national segregation. To effect this, freedom of trade, freedom of exchange, freedom of intercourse and personal freedom have to be rigorously suppressed. But although human beings in a community are the equivalent of the cells in an organism, they have achieved self-consciousness and individuality. If, therefore, the repressing force upon their originating and creative powers is strong enough these powers will die and degeneration will follow. *Laisser-faire* would lead to disintegration, and autocracy would lead to mental and moral slavery. The solution can only be found by a method which gives adequate freedom to the individual life within a larger co-ordinated unit. Can we achieve it? Perhaps we are neither wise enough nor good enough. It is no more reasonable to blame the present epoch for its savage and infantile psychology than it would be to blame the brontosaurus for its tiny brain. Has this civilization to go back to the melting-pot like its predecessors? If so another will rise in its place. Of that we may be sure, though it is little consolation to us or to our children. In the past, when a civilization has fallen, the rise of the next has been a matter, not of years, but of centuries, until some new dominant blend could arise. We are therefore naturally concerned to see the civilization of to-day conserved. If I could show the way, I might well claim the position of a beneficient dictator. All I am aiming at is to illustrate the truth of Maudsley's dictum: "The general law of development which governed the process during the unrecorded ages was the same law which is proved to have worked within the short compass of recorded time—namely the law of the more complex and special development at the cost of the more simple and general." In addition I hope I have analysed some of the defects inherent in that process. "The fault, dear Brutus, is in ourselves," rather than in a malignant fate obscurely moving behind the scenes. Can we not hope that by remorselessly stripping off the labels from outworn symbols, by resolutely adopting reality principles, we may, before it is too late, realize the latent possibilities in human life, and recognize that the springs of happiness come from within? In the willing co-operation of free individuals for the common weal lies the only solution. Is this a contradiction in terms? I believe not. We are members all of one body.

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PSYCHOSIS OR MALINGERING?

By FREDERICK DILLON, M.D.Edin.,

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THE subject of the following record was a married man, aged 40, whose business had brought in an income of about £2,000 per annum. The onset of his illness dated from an accident which was said to have occurred about seven months before he was seen by us. According to the history obtained from the wife it appeared that as he was passing a building in course of erection a fairly large piece of iron fell from the scaffolding and struck him on the left side of the head. He did not fall, but picked up the piece of iron, walked into the building and interviewed the foreman. He then went home, his only complaint being of headache. The wife said she noticed a swelling on the scalp, but as there was no bleeding or wound she did not send for a doctor immediately. The patient was put to bed and is said to have been sick on two occasions. As he did not improve, he was taken, after six days, to the out-patients' department of a general hospital, where the doctor who saw him attributed the condition to concussion. He was admitted four days later and remained for three weeks. The report from the hospital stated that the patient complained of headaches, became easily irritable and lost all power of control. He is said to have been sleepless, but no sickness had occurred. There was no mention of any external damage to the head, and X-ray examination of the skull revealed no injury. The blood Wassermann reaction was negative and the blood precipitation test was normal. No physical signs of disease were discovered. The wife stated that while in this hospital the patient chased one of the doctors down the stairs. Soon afterwards he ran away from the hospital, telephoned to his wife and was taken back by her, remaining a further week. He then returned home, and after a few days' rest resumed work. He was unable to continue as he became excitable and irritable, and on one occasion "smashed his office up". He was seen by his own doctor, who

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expressed the opinion that the accident had undoubtedly caused a hæmorrhage inside the skull, and that the clot so formed was responsible for the patient's mental disability. Ten days later he was examined by a distinguished neurologist, who also expressed the opinion that a blood tumour from bleeding within the membranes of the brain was the cause of the symptoms. The patient gradually lost interest in his work, stayed at home and spent most of the day in bed, preferring to be left alone. He became violent and on more than one occasion attacked his wife.

Thinking that a change might be beneficial, his wife took him abroad, about four months after the accident, and left him in a mental sanatorium. He remained there for nearly two months, but had to be brought back because of his violent outbursts. The day after his return he is said to have had a "fit", in which he lay stiff on the floor for about ten minutes, apparently unconscious. He was taken a second time to see the same neurologist, who then came to the conclusion that the patient was suffering from general paralysis of the insane. In this opinion the patient's own doctor and the insurance doctor concurred. As a result of this diagnosis, on the advice of his solicitor and of the family physician, the patient was taken to the observation ward of a local hospital, and from there transferred, after certification, to a county mental hospital, where he remained for about five weeks. The report accompanying him when transferred stated that no physical signs of any kind had been discovered. His blood Wassermann reaction was negative, and on investigation of the cerebro-spinal fluid the Wassermann reaction was found to be negative, the cells normal and Lange test normal. X-ray examination of the skull—the second carried out—disclosed no abnormality. In regard to his mental condition, he is described as having been admitted in a state of acute confusion, vaguely conscious of alteration in personality and mental capacity, with periods of gross amnesia, at times restless, agitated and hostile. The opinion was stated that the patient's condition was very suggestive of an organic lesion, of which a tumour was the most likely.

On admission to Northumberland House—about seven months after the accident—the patient appeared to be in a somewhat confused state of mind. He stated that he could not think clearly, and could not find words to express himself. He talked slowly, and his replies to questions seemed to be definitely delayed. He appeared to have an impaired power of recollection for recent events, and his memory for remote events seemed also to be defective. He was only able to give a fragmentary account of events during the previous few months, and orientation in time appeared to be affected. When questioned on the subject he declared that he had no recollection of an accident. He stated that he could not remember how he came to the hospital, and said that he felt quite a different person from what he used to be. Questioned on general topics he replied relevantly and was able to converse coherently. On physical examination the most striking feature was unsteadiness of gait, with a

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tendency to sway to the left. The pupils were large and equal, and reacted rather sluggishly to light and accommodation. He wore a belt for visceroptosis.

During the next ten days the patient's mental state showed no essential alteration, but he complained of numerous additional symptoms from time to time. These included localized headache in the left temporal region; fullness in the left half of the head and a feeling of relative emptiness and strangeness in the right half; pain in the back of the neck and in the neighbouring parts of the occipital region; pain in the left eye and a feeling as if it were pushed out; pain on pressure over an area in the left parietal and occipital region; dizziness from time to time, particularly on bending the head to the left; ringing in the ears; lack of "feeling" in his forehead and at the top of his nose. His gait continued to be characterized by a tendency to sway to the left and by the adoption of a wide base for standing and walking. His memory remained defective for recent and remote events. He said that he was puzzled as to how he got to the hospital and that he felt lost, as things did not seem to fit in. He appeared to become fatigued very easily, both physically and mentally; his powers of attention seemed impaired, there was difficulty in concentrating on any particular topic, and he took a long time to find words to express himself. He seemed somewhat depressed and anxious about his condition, and became rather irritable from time to time.

On the tenth day after admission a dramatic development occurred in the case. The wide range of symptoms, the incongruity of their combination, and their inconsistency, as shown, for example, by shrewd remarks made in a state of apparent confusion, had already aroused a suspicion that the question of malingering might have to be considered. On the day mentioned the patient asked to have an interview with all the doctors of the hospital in the presence of his wife, as he had something very important to communicate. This was not found to be practicable, but he was seen by both of us and made a statement to the following effect: He said that he had not been truthful in the description of his condition, that he had pretended to have symptoms which, in fact, he did not have, and that he had lied when he declared that he had no recollection of the accident. He gave an account of the accident, stating that he was walking along a street carrying his hat in his hand, and the next thing he remembered was a terrific knock on the left side of his head. He did not realize what had happened, but apparently did not lose consciousness or fall. He went into the building and spoke to the foreman. He had picked up the piece of metal which is said to have hit him and put it in his pocket. He said he had no idea at that time of claiming compensation; he felt dazed and stupid. After this he had no further recollection until he found himself in bed at home some time later. He could not say how he got home or what length of time elapsed. His recollection of subsequent events, he stated, was vague and indefinite. He felt, he said, that his personality had changed. He remembered telling lies about his condition, but could not help himself. He

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remembered the question of compensation being brought up and the sum of £250 being mentioned. He did not think this was sufficient, and persisted in his dissimulation because he wanted a sum which would be fair. He insisted that all he wanted was fair compensation. He declared that having once started to lie about his condition he found himself becoming involved more and more deeply until he could not see how to extricate himself. While in the county mental hospital he had tried twice to explain the situation to the medical officers but found he was unable to obtain a suitable opportunity. He declared that he was very worried about the whole matter now, and was anxious to get the certification if possible entirely annulled. He maintained that there were certain genuine symptoms present, such as headache, dizziness, and a feeling of falling to the right side (which was inconsistent with the objective sign of swaying to the left), and pain in the left instep of the foot which he attributed to the lumbar puncture performed on him.

This confession was made quite spontaneously and was given in a clear and connected manner, with the accompaniment of a good deal of apparently genuine emotion—tears rolling down his cheeks on more than one occasion. During the next few days, however, no obvious change took place in the patient's condition. He made a few interesting remarks such as—" I have studied a little psychology and there is a little resistance in me ", " My income tax is the hardest task in my business ", and " I have always the idea that I am getting worse and worse ", but he did not act as if the assumed mental disorder had cleared away. A few days after the interview he said that he did not remember the accident but that he remembered being told about it, and later he stated that he had seen another man involved in an accident and that he (the patient) had helped him. He added that people forced him to tell lies.

In view of these discrepant remarks he was interviewed again seven days after his confession. He then said that he had no recollection of the previous interview, and added that everything appeared to be mysterious—for example, why he was at Northumberland House. He denied ever having had an accident, and when asked if he remembered any conversations about an accident, answered rather vaguely, " Maybe; I think I have been influenced to a certain extent by other people ". From previous remarks it appeared that he was referring to his wife. Two days after this interview he became more restless, irritable and excitable and on occasions violent, and in one of these phases he broke a window. He stated that he felt inclined to hit people but did not know what it was that irritated him. Several times he had to be given hyoscine because of his instability, and after a struggle with the nurses on one occasion he complained that one of them had hit him on the stomach with a slipper—a statement for which no corroboration could be found. A few days later the patient was discovered lying in bed with his pyjama trousers tied tightly round his neck, and on the nurse attempting to

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undo the knot the patient resisted and tried to prevent him. He became violent, smashed a flower vase, and again required to be given a sedative. On waking he appeared confused and depressed, said he wanted to commit suicide, and asked to be given something "really strong" that would do away with him, as his headache was agonizing. Next day he seemed much clearer in his mind, talked in a more connected and coherent way and asked for the first time to be allowed to go home. He said he was not insane, and that he could think clearly but had a difficulty in getting his ideas together.

Another well-known neurologist who examined the patient at this stage—about a month after admission—stated that in his opinion there were no signs of any organic lesion, traumatic or otherwise, in the central or peripheral nervous system. In the course of the next few days the patient stressed the fact that he thought another man had had the accident and that he, the patient, wanted the other man to have the insurance money. He added that at times he was not quite sure whether he was the other man or not, and said, "I have nothing to do with the accident. I think somebody has influenced me to say I have had the accident". It was felt that he should be given the opportunity to get out of a difficult psychological situation and he was asked frankly if he were pretending to be confused, to which he replied that he was not. Questioned about the confession he had made he said he did not remember it. Later, however, in the same day he said he did remember an interview, and declared that somebody had prompted him to state that he had had an accident. He added that a man who camouflages has to have patience, and he had no patience. "Supposing I hadn't had an accident," he said, "I would be a poor man"—which he corrected immediately to "Supposing I had an accident I would be a poor man". Later he remarked, "How could a man for a lousy £750 make a mess of his business affairs?" Some hours later in the same day he appeared very depressed and said he felt convinced that he was insane. The following day when the medical officer entered the patient's room he sprang up from his chair and raised his arms as if to strike, shouting, "Get out; I heard you call me a liar". His wife, who was present, intervened and was able to calm him down.

About this time he began to act as if he were influenced by auditory hallucinations, adopting a threatening attitude on occasions, and accusing different persons of saying he was a liar and that he didn't want to work. A day or two afterwards his condition became worse. He became more restless and confused and refused food. About 5.30 one morning he appeared to be in a state of collapse; the pulse was weak, the temperature was 95° F., his articulation being thick, slurring and hardly comprehensible. Later in the same morning two tablets were discovered in a drawer in his room. Questioned as to how he had obtained them, he gave a confused account of having been given them by a doctor while he was on the Continent, and admitted having swallowed two of them. His explanation was obviously fictitious.

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It was impossible for him to have had the tablets concealed as he suggested, and a new turn was given to the situation when it became known that the wife had visited the patient the day previously. The tablets were analysed and found not to contain any sedative drug, but an organic compound of sodium and arsenic. A fresh significance appeared when it was discovered later that the patient thought the tablets consisted of medinal.

This acute phase of confusion appeared, so far as could be judged, to be genuine ; it lasted about forty-eight hours, but he remained apparently dazed, irritable and hesitant in speech for about a week. After this he became clear and rational and continued so for about ten days. He then remarked that he felt a change in his personality, and thought that he might be leading a double life. He added that the question of compensation should not be brought up too often, but that his case should be proceeded with on a new basis of bringing the family together. This last remark became significant in the light of information that was obtained a little later about his wife. The following day he said that he wanted to make a statement to the doctor. He then made a detailed confession in which he admitted that not only had he been pretending most of his symptoms while in Northumberland House, but had been simulating right from the beginning. He declared that he had no recollection of an accident, and that his memory of the few days afterwards, before he went to the first hospital, was vague and indistinct. He remembered being worried about business affairs, which were not progressing well, learning that he had been diagnosed a case of concussion, and hearing that a sum of £250 had been mentioned as compensation. He felt most indignant at the insignificance of the sum, and admitted that after he had left the first hospital he bought some secondhand medical books and carefully read up the subject of concussion with a view to making his demonstrations more convincing. Before every interview with a solicitor or doctor he took large doses of medinal, and he admitted that on the occasion of the " fit " and when the diagnosis of general paralysis had been made he was under the influence of the drug. On two or three occasions, he stated, while in Northumberland House he had produced symptoms by the use of medinal. He was reticent when asked how he had obtained the drug, but his remarks left little doubt that it had been brought to him by his wife. He had " acted " hearing " voices ", he said, and his condition while in the county mental hospital was pretended " from *a* to *z* ". He had staged a period of violence there because he thought they were going to discharge him. His one desire, he added, was now to re-establish himself as a sane and honest man and he thought his only hope was to abandon all claims for compensation.

After this confession the patient remained in the same clear and rational state of mind. He complained of headache, dizziness, some blurring of vision and of sleeplessness, for which he was given a mild sedative, but there was no return of the psychotic type of symptoms. It became evident in the course

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of further conversations that he had been considerably troubled over business and domestic affairs, and it was worry over the latter that apparently was responsible for clearing up the syndrome. He had become suspicious that his wife had formed a friendship with another man and was losing interest in him. This he later found to be the case, and discovered that his wife was, in fact, contemplating divorce. He thought also that she was arranging to send him out of the country—which would have been ruinous to his means of livelihood and future prospects. These anxieties seemed to have acted as the stimulus which led him to make his final confession.

The patient was kept under observation for a further period of three weeks, during which time he remained normal, clear in mind, lucid in conversation and consistent in the account he gave of himself—which we were able to verify in a number of particulars. He reaffirmed his decision not to pursue the action for compensation. His wife and his solicitor were informed of the development that had taken place in the case, and by the former's instructions the patient was discharged from certificates and left the hospital, 2½ months after admission. We heard on two or three occasions after this from both the patient and the wife, and it was evident that considerable dissension was taking place between them. But three further episodes complete the record of the case. The first was that nine days after his discharge the patient came to see one of us and said that he had changed his mind and now intended to continue with the action for compensation. He stated that one of the reasons was that he was in debt and needed the money. He admitted the malingering, but declared that as the result of the accident he suffered from a number of symptoms such as headache, periods of confusion, impairment of memory and loss of concentration. He had, he said, been examined by another doctor, who had informed his wife that he was suffering from " progressive paralysis ". He wanted to be reassured on this point. The second development consisted of a communication about six months later from the man's solicitor to the effect that the patient had been examined jointly by a psychiatrist and neurologist—both very distinguished in their respective spheres—and as the result of the serious report they gave of the patient's condition and future prospects the insurance company settled the matter out of court by awarding the patient a sum of money amounting to nearly £2,000. The third episode occurred about a month later when the patient again visited one of us and gave further details of the award which had been made. He admitted frankly that he had " taken in " the neurologist and psychiatrist whom he had last seen, and on whose report the award had been made. He indicated that he was having a " good time "—" enjoying good food and good wine ". His physical condition had improved and he had put on weight. The domestic difference had apparently been settled ; he was living apart from his wife and was on the look-out for a new partner with whom to commence business again.

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This is an unusual case, but it serves to show the possibilities that are open to a clever and determined person. Few people after reading the foregoing account will doubt that the man was a malingerer. At the same time it has to be admitted that scientific proof is lacking, as there are no tests which enable one to distinguish with accuracy a psychosis from a simulated condition. Verification is made more difficult by the fact that the man could, without any doubt, have been able to produce at will a "relapse" of the symptoms which in all probability would have satisfied a court of law. It is generally held that insanity is rarely feigned, and that the majority of cases described as such in earlier records were errors of diagnosis. Persons who simulate psychotic conditions are said either to be predisposed to these disorders or to have previously suffered from insanity.* There is probably a good deal of truth in this statement—at least if we substitute the wider concept of mental abnormality for psychosis. When we remember also the advance made in the study of abnormal psychology in recent years, and the greater facility this has given us of diagnosing abnormal mental states, the statement gains additional weight. In regard to the case we have described, the limiting possibilities are that it was a case of pure malingering on the one hand or, on the other, a psychosis brought about by injury to the skull. Absolute malingering we may eliminate from consideration as there is no likelihood of ever obtaining accurate evidence on this point. The alternative of severe injury was the one accepted by all the other doctors who were associated with the case. On this point it is interesting to note that no external mark of injury was ever discovered by any of the doctors who examined the patient, and no damage to the skull was found by the X-ray investigation—which was carried out on two separate occasions. The only evidence that an injury had ever been sustained came from the man's own report, and from the statement of the wife that she had noticed a swelling on the scalp. It was significant, however, that she did not obtain medical advice until six days had elapsed after the accident. From the patient's description of the size and weight of the piece of metal that is alleged to have hit him it is incredible that it could have done so without at least producing a wound on the scalp—probably a considerable lacerated tear with much bleeding—especially as his hat was not on his head at the time. It is a distinct probability that no injury ever occurred on the occasion in question, and the very self-possessed action of the patient in walking into the building and interviewing the foreman, in spite of the "terrific knock" he said he had sustained, lends support to this view. It is further significant that his recovery was rapid, if not immediate, after compensation had been paid, although the amount of this had been decided partly upon the assumed pathology and the bad prognosis that had been given at the final examination of the patient.

Apart from pure malingering and serious injury, however, there are other

* *Malingering*, A. Bassett Jones and Ll. J. Llewellyn, 1917.

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possibilities that fall to be considered. If we assume that a slight injury did occur, the case might then be similar to a traumatic neurosis in which unconscious exaggeration of symptoms takes place. On the other hand, it is easily credible that deliberate exaggeration of symptoms arising from such an injury may have been carried out—a syndrome which would require to be distinguished both from pure malingering and genuine traumatic neurosis (or psychosis). In this connection it is interesting to note that there is evidence to support the view that a genuinely abnormal state of mind may come to be developed in this way. Collie remarks* that the simulation of melancholia may actually bring about the condition that is being feigned. Another alternative is that the injury, if admitted, may have brought about an alteration of personality, as sometimes happens in such cases. Some plausibility is given to this theory by remarks of the patient to the effect that he felt a different person, that it was another man who had had the accident, that he seemed to have a double personality, and that he told lies about his condition but could not help himself. This attitude, however, was far from being consistent, and the fact also that the patient admitted he had obtained books and read up the subject and actually confessed that he used the expressions mentioned above to deceive the doctor—to say nothing of his subsequent rapid recovery—enables this hypothesis to be discounted. Finally there is the possibility that we have to deal with a psychopathic personality apart altogether from the question of an accident. There was no record in the personal history we obtained of any previous nervous or mental disorder, but the only source of information open to us was the patient's wife, whom we did not look upon as reliable. In all probability adequate knowledge of the patient's history and personality would disclose the existence of psychopathic traits. But unless we assume simply that the man was an exceptionally clever rogue willing to go to great lengths to achieve his ends, the evidence strongly points to the fact, no matter which of the diagnostic possibilities we may accept, that a considerable element of malingering was present in the case. A definite and adequate motive existed. The patient was in financial difficulties, and his references to compensation showed that the subject was frequently if not constantly in his mind. The evidence of injury was remarkably inconclusive; the features of the mental disorder were inconsistent. The patient confessed to taking medinal to produce symptoms and admitted reading up the whole subject. These facts together with the clearing up of the symptoms after compensation had been paid and the confessions he made all go to make this conclusion obvious. A confession in itself is not, of course, decisive evidence of guilt. It may be the result of an abnormal state of mind, as are many confessions of crimes, for example, that are made to the police. Or it may be only partly true, and in this case it is probable that his admissions were never frank and honest. His first

* *Fraud in Medico-Legal Practice*, Sir John Collie, 1932, p. 119.

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confession was probably a "try-out" to see how it would be taken by the doctors. It seemed to be a fact that he had become involved in his deception more deeply than he anticipated—not many persons would go so far in simulation as to consent to be certified and sent as a rate-aided patient to a mental hospital—and could not see the way out. He had also become alarmed at the attitude of his wife. There is little doubt that she was assisting him in the deception, but she had formed a friendship with another man, wanted a divorce and contemplated sending him out of England. As she was the breadwinner and he only her manager it was an important development for him and formed the incentive for his "recovery," to enable his discharge from certificates to be obtained. The case illustrates the care that needs to be exercised where a compensation motive is concerned.

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THERAPEUTIC NARCOSIS WITH SONERYL*

By J. Stephen Horsley, M.R.C.S.,
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Barbital therapy has aroused more interest than any other method of narcotic treatment. The reason is that barbital narcosis offers a new means of establishing contact with hitherto suspicious, seclusive and inaccessible patients. It is the quickest and most effective method of inducing sleep and rest in various maniacal states. Furthermore it is a potent agent in terminating the course of many acute psychoses. Barbital therapy, being empirical, and to some extent experimental, is difficult to define. It denotes more than the mere induction of continuous deep sleep. In fact, it denotes an organized scheme whereby physical and psychic rest, increased suggestibility and alteration of affect are used together to restore normality.

In the absence of precise knowledge of the mode of action of barbituric acid the choice of its derivations depends to some extent on personal preference. The opinion at Herrison based on 40 prolonged narcoses with soneryl is that the drug is less toxic and sometimes more effective than somnifaine.

Pharmacology of Soneryl.

Soneryl is n-butyl-ethyl-malonylurea, a "heavy barbiturate" comparable with nembutal, amytal and dial. It is prepared in several forms to suit the varying requirements of administration. The effects are similar. Small doses in psychotics produce anomalous emotional states, with a tendency to become either confiding or lachrymose. A full therapeutic dose produces deep narcosis lasting rather longer than that due to nembutal. Slight cyanosis is common, but there is no change in the rate or rhythm of respiration. An initial quickening of the pulse is followed by definite bradycardia. Simultaneously the blood-pressure falls by about 25 mm. of mercury. The cerebro-spinal fluid pressure falls abruptly and tends to remain subnormal throughout narcosis. Over-dosage produces great depression of all vital processes. A significant and early sign of poisoning is a soft, slow pulse of about 40. This may be followed by collapse, with subnormal temperature, dilated pupils and a dusky complexion. The most effective antidote is coramine in doses of 5 or even 10 c.c.

* Paper read at the Autumn Meeting of the South-Western Division of the Royal Medico-Psychological Association, 1936.

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Administration and Dosage.

The arrangement of a practicable technique was preceded by a series of test doses in which various methods were applied to separate groups of 50 patients. The first method of oral medication, basing the dose on body-weight, showed partial or complete failure in 10% of cases and was abandoned. The second group received the salt, sodium soneryl, by intravenous injection, and here it was considered safer and more accurate to regulate the dose by individual response, i.e., by inducing narcosis slowly and stopping the injection as unconsciousness supervened. This method never failed to induce sleep, and in single doses had no adverse effect on the circulatory or respiratory systems. Moreover the increased restlessness which occurs on waking in 10% of cases after oral medication was not seen after intravenous injection.

The third group received soneryl sodium by intramuscular injection. The narcotic effect was apparent in 5 or 10 minutes, and its duration equalled that due to intravenous injection. Unfortunately, the solution is a local irritant, and 4 cases developed deep-seated injection abscesses. An alternative preparation of soneryl, in special solvent, has been used in 20 cases without ill-effect.

The fourth group received suppositories in doses of 5, 10 and 15 gr. These were given according to a scheme which allows for possible idiosyncrasy. This method was invariably effective and well tolerated, thereby resembling the highly satisfactory results already recorded with nembutal suppositories (1).

The conclusion is that the intravenous and rectal methods offer the greatest therapeutic efficiency. These methods will be described separately and in detail.

Part I.—Intravenous Therapy.

The immediate and profound effect of intravenous narcosis is of incomparable value in psychiatric emergencies. Objections have been raised that intravenous narcosis introduces the risk of sudden and irrevocable toxic effects, whereas the truth is that the slow injection of a dilute solution is the safest and most accurate method of estimating the correct dose. The danger of thrombosis, which has been described with somnifaine, is negligible with some of the newer barbiturates. We have not seen this complication in over 3,000 intravenous injections with amytal, nembutal and soneryl.

The value of these drugs in suicidal cut-throat is obvious, and their use in status epilepticus is well known. Their use in acute delirium is not so well defined. The difficulty here is to maintain narcosis for a period long enough to be curative. The most able exponents of somnifaine therapy will admit that in certain cases it is impossible to induce a satisfactory narcosis. Ström-Olsen (2) says that " in such instances it is advisable to cut short the

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treatment rather than to continue injecting large quantities of the drug". The present purpose is to suggest an alternative method of treatment for those acutely delirious and toxic cases whose feverish over-activity so often ends in exhaustion and death. In treating such patients rest alone is insufficient, and it is necessary to take active measures to eliminate the toxæmia. In order to do this effectively, we turn to the surgical procedure of continuous intravenous infusion of saline.

Continuous Intravenous Narcosis.

The continuous infusion of saline by the intravenous route is established as a life-saving procedure in various surgical emergencies. An excellent account of the technique was given by Hamilton Bailey (4), who described the value of giving intravenous saline continuously for a period of 3 to 5 days. He also indicated the value of giving simultaneous intravenous medication—the appropriate drug being added to the saline, which can be infused and regulated with mathematical precision. Clearly in the treatment of the acute toxic psychoses it is equally rational to give prolonged infusions of glucose saline, adding as required minimal doses of the appropriate narcotic.

Infusion Technique.—The technique described is a modification of Bailey's (4). We assume that the general principles are known. Either a gold-plated cannula or a special needle* may be used. The advantage of the special needle is that it is quicker and easier to insert in a resistive patient.

The choice of vein is a question of convenience, and it is found equally satisfactory to use the back of the hand, the front of the forearm, or the lower extremity. The limb is kept at rest by means of a light anterior splint in the case of the hand, or a Thomas knee-splint in the case of the lower limb. In applying the splint it is necessary to take care to avoid any proximal pressure liable to obstruct the vein.

The solution.—The most useful solution is 5% glucose in normal saline, and it is recommended that 10 units of insulin be added to every 500 c.c. of glucose solution. Several methods are advocated for regulating the temperature of the fluid. This, however, is relatively unimportant, and it is far better to keep the patient warm with extra blankets and hot bottles.

Rate of flow.—The rate of flow is regulated by a thumbscrew to about 30 drops per minute. The narcotic is injected into the tubing just proximal to the needle, and this is repeated in minimal doses whenever the patient becomes restless. There is no fixed rule for determining the maximum dosage or the maximum duration of infusion. These must be decided entirely by clinical experience, together with the general condition of the patient. In our experience it has been necessary in severe cases to inject as much as

* The needle is of carbon steel gilt and is made by Down Bros., Ltd.

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3 grm. of soneryl during the first day. This somewhat excessive dosage seems to be made safe by its extreme dilution and very slow administration in saline. The infusion may be continued in most cases with reasonable safety for 48 hours, by which time it is possible to revert to ordinary methods of treatment.

Special precautions.—Infusion narcosis necessitates the constant attention of a nurse, who for preference should have special experience of this treatment. She should be competent to measure and record the blood-pressure. It is her duty to regulate the rate of infusion, and to inject the repeated doses of soneryl whenever necessary. For her guidance she is given the following printed instructions:

1. The patient must be under constant observation.
2. Chart the pulse and respiration every 15 minutes and the temperature and blood-pressure every 4 hours.
3. Regulate rate of infusion to between 20 and 30 drops per minute.
4. Look for swelling under the eyes or in the limbs.

Important.—The nurse must report *at once*:

(i) Pallor or cyanosis.
(ii) Pulse-rate below 45 or above 90.
(iii) Any respiratory distress.

The following examples indicate the great value of this method in the treatment of very ill patients:

CASE 1: *Married woman, aged 24. Puerperal confusional state.*—There was no personal or family history of epilepsy or psychosis. The present illness was of sudden onset on the fourth day of the puerperium. On admission there were signs of severe toxæmia, the tongue was dry and furred, with sordes on the lips. There was also pyrexia, tachycardia, albuminuria and moderately severe ketosis. The mental state was one of excited confusion, the patient persistently fighting, biting, shouting and exposing herself shamelessly. She had refused all food for several days. During the first two days ordinary methods of sedation and tube-feeding completely failed to effect any improvement, in fact she was becoming more ill. Continuous infusion of saline was then begun, together with an initial dose of 1 grm. of soneryl sodium. During the following 24 hours the infusion was continued at the rate of 30 drops per minute, together with 2 c.c. of 10% soneryl approximately every hour.

By this time her general condition showed signs of improvement and the infusion was discontinued for 2 hours, when she once more became noisy and restless. The infusion was resumed and continued uninterruptedly for a further period of 48 hours. During this time she received a further quantity of 2·8 grm. of soneryl sodium. By now almost all signs of toxæmia had disappeared. Her mouth was moist and fairly clean and the ketosis had ceased. She was allowed to wake from the narcosis, and was found to be sufficiently manageable to be nursed in the open dormitory. She was still mildly restless and confused, with delusions of identity. Four days later she had a major epileptiform convulsion, following which there was a prompt change in her mental condition. She began to realize her circumstances and to be more co-operative. The remainder of her illness was uneventful, and

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ran a more or less typical course. The immediate benefit to her mental condition was only slight, but in the relief of her exhaustion and toxæmia it may well have been a life-saving procedure.

CASE 2: *Married woman, aged* 39. *Acute delirium.*—On admission her illness had lasted about a month. She looked ill; her tongue was thickly coated and dry. There was a moderate degree of albuminuria, and her blood-pressure was 135/90. Her mental state was one of gross confusion. She was disorientated, noisy, incoherent and violent. Visual hallucinations were of an intensity recalling delirium tremens. She saw spiders everywhere and this increased her noisy excitement. The usual treatment, including the use of hypnotics, sedative baths and nasal feeding, failed to effect any improvement. She was given a continuous infusion of glucose saline. The infusion was continued uninterruptedly for 62¼ hours; the total quantity of soneryl was 3·1 grm., most of which was given during the first day. By the third day she was coming round from the narcosis but was no longer resistive. Her mental state had changed completely to one of slight depression. When the infusion was discontinued she showed signs of returning restlessness, but after 3 days she was clear, amenable and rational. Convalescence began from this moment, and a week later she was moved to an open ward, where she stayed about 6 weeks before being discharged. She has kept well.

Continuous intravenous narcosis is drastic but effective. It is regarded solely as an emergency measure applicable to very ill patients who are failing to respond to the more usual methods. So far we have treated 10 patients by this method with encouraging results. Nine showed immediate improvement, but the tenth, who was very ill, failed to respond, and died. In concluding this preliminary account of infusion narcosis we would suggest that it offers a possible means of treating acute psychoses in the wards of a general hospital.

Part II.—Prolonged Narcosis with Soneryl Suppositories.

The general management is based on the well-known technique of the Cardiff school (3). The patient is examined, prepared, and put to bed in a quiet side-room. The usual arrangements are made for daily laboratory examination. The principal modification is the substitution of soneryl for somnifaine with consequent advantage of simplicity in administration. Many exponents of somnifaine therapy aim at producing a deep sleep from the very beginning of treatment. This method fails to allow for possible idiosyncrasy, and is not without danger. We prefer the method advocated by Meerloo (5) of using the first three days as a test period to determine the individual reaction of each patient.

The initial narcosis should be controlled by doses which do not exceed 5 gr. every 6 hours. It is often sufficient to give 5 gr. twice or thrice daily. The most important feature during the test period is the condition of the pulse, a pulse-rate below 50 being the commonest sign of intolerance.

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After 3 days, subject to the general condition being satisfactory, it is safe to increase the dosage. The maximum single dose is 15 gr., but 10 gr. repeated two or three times a day is usually adequate for producing a fairly continuous and deep sleep. In connection with dosage, it is advisable to consider the routine administration of insulin. Glucose, being an essential ingredient of a nutritious fluid diet, is given as a matter of course. The addition of insulin is believed to raise the tolerance to soneryl and to necessitate larger doses. Therefore insulin is reserved for cases showing ketosis. Slight acetonuria occurred in about half the present series, but it was never severe, and always disappeared on giving insulin, glucose and copious fluids.

The whole problem of dosage is empirical and uncertain. To give fixed doses at regular intervals is to court disaster ; in fact, the only safe method is to regulate narcosis day by day according to the general condition of the patient. Some patients are managed easily and sleep almost continuously with the smallest doses ; others respond only to large and frequent doses. The ideal is to produce continuous deep sleep, unaccompanied by toxic effects. This is seldom achieved, and yet, in cases where sleep is by no means continuous, the results are almost as good. A study of such cases shows that although the drug does not necessarily produce sleep, it invariably produces other effects described collectively as " barbital intoxication ". In such cases the effects of narcosis may be paradoxical, consisting of acute hallucinatory excitement and motor restlessness throughout the period of intoxication, followed by sudden complete recovery a few days after abstention. As Meerloo (6) says " sudden disintoxication is an important factor and sleep constitutes no very real element ". The following case illustrates the paradoxical effect of soneryl with subsequent recovery in schizophrenia :

CASE 11 : *Single female, aged* 19. *Schizophrenia.*—There is no personal or family history of epilepsy or of previous mental disorder. The present illness began with sullen behaviour, laziness and complete indifference towards the future. She caused her family great anxiety by wandering away from home in an aimless manner, sometimes being brought back by the police. On one occasion she said she had been out " seeking death ". There was no amnesia after these flights to suggest epilepsy. On the contrary, they appeared to be typical wanderings of a schizophrene, as she failed to appreciate the alarm she caused.

She was admitted as a voluntary patient, and was aloof, autistic, and absorbed in phantasy. There was no evidence of hallucinations or delusions, but she was puerile and apathetic, and would not be employed. She was given a course of soneryl for 12 days in three phases :

Phase 1 lasted about 3 days, and was characterized by acute confusion, delirium, and persistent attempts at self-injury, chiefly by banging her head against the wall.

Phase 2 was characterized by catalepsy, dramatic posturing and vivid hallucinations of vision and hearing—she heard the voices of both her parents calling to her, and she saw the face of her mother with the wings of an angel hovering over her.

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Phase 3 was continued with the same dose of 30 gr. daily. She became calm and restful, but did not sleep—in fact, she appeared to have developed some degree of tolerance to soneryl. Hallucinations had faded, and she was able to discuss them but with some perplexity.

Narcosis was discontinued, and on the following day she became emotional and lachrymose. The next day she had a major epileptic fit with unconsciousness, cyanosis and tongue-biting. Immediate examination failed to reveal any other abnormal neurological sign. Nevertheless there was a transient amaurosis. The accompaniments of the fit were interesting; she complained of the most intolerable burning headache, unlike any she had ever felt before. The pain was in the region of the vertex, lasting all day and accompanied by persistent cerebral vomiting. Towards the evening she was given an intravenous injection of 50 c.c. of distilled water. The headache and vomiting ceased but her temperature rose suddenly to 100°. The following morning there was slight headache, but she was otherwise clear, coherent and apparently normal. This was followed by satisfactory convalescence, willing interest in occupation and apparent complete return to normality.

Duration of Narcosis.

The optimal duration of narcosis is quite uncertain, although the general consensus of opinion is that a period of not less than 10 days is required. In the absence of definite criteria we prolong most narcoses for an arbitrary period of 14 days. Nevertheless, in cases with toxic complications and consequently abbreviated treatment, the results were equally good. The following example illustrates the relief of a melancholic attack by 5 days' narcosis:

Case 19: *Single woman, aged* 39. *Melancholia.*—The personal and family history were negative. On admission the patient was persistently depressed and agitated, reiterating her wish to be allowed to die or go at once and give herself up to the police. She was very resistive to all attention and difficult with food, ultimately needing to be tube-fed. She was given a course of treatment for 5 days, with an average dose of gr. v *b.d.* During this time she took food voluntarily and in fair quantity. In spite of other conditions being satisfactory her pulse became slow and feeble. On the third day it was 42, and by the fifth day had reached the exceptionally slow rate of 32; for this reason narcosis was stopped.

The following day the patient was much brighter, although still mildly depressed and a little retarded. Her appetite was good, and she showed interest in therapeutic occupations.

There is another side to the question of duration. Certain writers have referred to very prolonged periods extending over several weeks, or even months. Their reports of success in seemingly hopeless cases appear to justify this unusual procedure. A possible objection to very prolonged narcoses is the greater incidence of secondary effects, such as epilepsy. Some of the commoner by-effects are now considered.

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Secondary Effects.

(1) *Cravings.*

The importance of the apparently unrelated secondary effects of barbital intoxication is that they are of value in interpreting psychopathological processes. The commonest psychic phenomenon of soneryl intoxication is a persistent and regularly occurring craving for food and drink. This craving has been observed in both circular and schizophrenic psychoses, but the most noteworthy feature is its consistent appearance in melancholia. In its regularity, simplicity and persistence, this craving in melancholia suggests a regression to the infantile level. The patient wakes at regular intervals for meals, makes insistent requests for food, and then on being fed sleeps soundly until the next meal-time. This phenomenon has appeared in seven consecutive cases of melancholia, and has been of practical value in improving the physical condition. A point of interest was that each patient had a fixed phraseology, which she repeated at every meal-time. The following are actual examples :

Case 30.—" Please give me food and drink ; please give me food and drink."

Case 31.—" I want bread and cheese."

Case 32.—" I'm hungry ; I should like rice pudding ; please give me rice pudding."

Case 33.—" I do want some fruit ; can I just have an apple."

Case 34.—" I want a lemon drink, then I'll go to sleep."

Case 35.—" Do bring me a cup of tea and something to eat."

(2) *Catatonia.*

The relation of soneryl narcosis to catatonia is doubly interesting : on the one hand prolonged intoxication may produce a transient catatonic syndrome where it is least expected, namely in the manic-depressive states ; on the other hand true catatonic schizophrenia is relieved. It seems advantageous first to consider the more easily explained effects in schizophrenia. The effects described are not peculiar to soneryl, but have been observed also during medication with various barbiturates. The most relevant reports are those concerning sodium amytal, which was used by Bleckwenn (7) to induce spontaneous speech and lucid intervals in protracted cases of catatonic schizophrenia. Thorner (8) observed the same effect, and ascribed it to the inhibition of higher inhibitory centres. This mechanism is believed to explain the action of soneryl in those cases where it is effective in interrupting the negativism of catatonia.

Ten cases of catatonic stupor were chosen on account of their long chronicity, degradation, mutism and apparent dementia. The effect of narcosis was remarkable in that four of them not only spoke spontaneously and rationally, but showed by their conversation that apperception had been present and

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memory preserved throughout the long period of stupor. The other six patients, who appeared to be hopelessly demented, did not improve ; nevertheless three of them who for many years had shown no sign of emotion, wept repeatedly during narcosis. The following case illustrates the probable mechanism of narcosis in catatonic stupor :

CASE 20 : *Single woman, aged 24. Catatonic schizophrenia superimposed on congenital mental deficiency.*—The history was incomplete, but there was feeble-mindedness in a sister. The patient showed signs of backwardness at school and only reached Standard 2, but she did not become certifiable until the age of nineteen. The psychosis began with dislike for work, she became sullen, depressed and excited, and threatened to hang herself. On admission to hospital she was in a state of catatonic excitement. This continued for some months, and was followed by a long period of stupor, during which she was tube-fed. During the following four years she appeared to be quite untrainable, and a continuing report at this time stated " she is oblivious to her circumstances and quite inaccessible. She does not speak or take any notice when spoken to ; she is faulty in habits, and has to be fed and looked after in every respect ". There was no apparent change for a further twelve months, when she was given a course of soneryl. During narcosis her expressionless immobility changed to one of wistfulness, with spontaneous conversation about herself. She referred to her stupor as " dreaming ", and said that she did this to get away from her work, where she had been unhappy. She said that people had laughed at her because she was not a good scholar, and that she did not want to return to her home, where she would have to work. Her explanation shows her willing escape into schizophrenia. The improvement was only temporary, and soon after treatment she relapsed into her former state of stupor.

The paradoxical phenomenon of induced catatonia is difficult to explain. It may appear early, persist throughout narcosis, and disappear suddenly on disintoxication. On the other hand, it may appear as a transient feature, alternating with complete passivity and catalepsy. The statuesque attitudes and spastic immobility of schizophrenia occur also in the induced catatonia of narcosis, but here the resemblance ends. The distinction becomes apparent in the lucid intervals after treatment when the melancholic is found to have been dominated by strong emotions, whereas the schizophrene is not. The catatonia of the melancholic is a dramatization of her affective state, that of the schizophrene being purposeless and autistic. The following case illustrates the sequence of cravings, desires, catatonia and catalepsy during the course of seventeen days' narcosis :

CASE 30 : *Married woman, aged 40. Agitated melancholia.*—It is believed that her father attempted suicide. The patient's history appears to have been normal and uneventful until February, 1936, when she became depressed, irritable and emotionally unstable. She was treated in a nursing-home for three months but did not improve, until finally and unexpectedly she jumped through a window and fell to the ground 40 ft. below. There was no evidence of serious injury, although she was very severely bruised. She was certified

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and admitted to hospital in May. Her mental state was one of agitated depression, in which she was unable to give any clear account of herself, but would only repeat phrases to the effect that she was very wicked, but too wicked to die. At times she was intensely preoccupied, listening to hallucinatory voices which told her that she was Satan. Her expression and attitude were always those of extreme misery. She was quite unemployable, would not eat and could not sleep. The usual sedatives were of little benefit, and although her physical condition was very poor and rapidly deteriorating, it was decided as a last measure to attempt prolonged narcosis.

The first day she received two doses of 5 gr. and slept most of the time. By the second day the symptom of food-craving had appeared and her whole appearance had changed. At each meal-time she sat straight up with a mask-like face and eyes tightly closed, pinching her nostrils all the while with the finger and thumb of her right hand. She asked for food and drink, but would say nothing else ; nevertheless, she ate readily and with a peculiarly slow and deliberate munching. The next phase in her narcosis was characterized by erotic desires towards the doctor and towards one of the nurses. She would not speak to anybody else, however, and within a few days the third phase appeared. This was one of catatonia. She knelt in an uncomfortable position and appeared to be praying, although at the time it was impossible to determine the nature of her thought or feeling. Phase 4 showed a complete change, with extreme docility, quiet amiable speech, but considerable apathy and the phenomenon of flexibilitas cerea. The fifth and last phase of narcosis was a return to catatonic stupor with impulsive conduct. She became somewhat difficult to manage and therefore the narcosis was discontinued.

There was little change in her mental condition until five days later, when a sudden change appeared. She began to talk in an abrupt, inhibited style, speaking of her wish to get up and return to her home. The nurses reported that she was brighter but very impulsive, making several sudden rushes towards the door. Her condition for several days was one of depressive mania, followed by gradual convalescence.

(3) *Post-narcotic Epilepsy.*

Many writers have described isolated instances of epileptiform convulsions occurring during, or more often after treatment with basal narcotics. These fits have been observed in most cases a few days after the cessation of treatment, and the inference is that they are due to sudden withdrawal of an anti-convulsant drug. Meerloo (5) describes 5 cases of epileptiform reaction in 500 somnifaine treatments, and a sixth case of a patient suffering from veronalism who later died in status epilepticus. Meerloo offers the interesting theory of a normal though latent " Krampfbereitschaft ", the convulsive discharge following sudden abstention from the narcotic. Kooy (9) describes two instances of epileptic fits in a series of 130 somnifaine treatments, and he suggests the theory that " many individuals have a latent tendency to epilepsy ". Ström-Olsen's (2) suggestion that the fits are causally related to hyperæmia of the brain is less easy to correlate with the fact that they occur more often after than during the treatment. A fourth and more acceptable suggestion is

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MacNiven's (10). In his series of 40 treatments with somnifaine he observed symptoms of cerebral irritation in 3 cases. " In one of these the temperature rose to 103° on the last day of treatment. The patient developed symptoms of irritation with marked opisthotonos. This symptom lasted for three days and then subsided completely." The second case developed symptoms the day after treatment was terminated. " The patient had four epileptiform convulsions in the next twenty-four hours. Her symptoms suggested calcium deficiency, and she was treated with calcium by the mouth. Her symptoms subsided completely." The third case developed tremors of the face and limbs, which subsided following treatment with calcium gluconate.

There are indications suggesting that tetany rather than epilepsy is the correct description of post-narcotic convulsions. If this is so it is necessary to explain why the convulsions occur in most cases after cessation of narcosis. The inference is that lowered serum calcium produces a convulsive tendency, temporarily checked, but becoming apparent on sudden withdrawal of the narcotic. Further investigations are necessary to correlate the convulsive tendency with the parathyroid dysfunction suggested by Hennelly and Yates (11).

The reported incidence of post-narcotic epilepsy is about 1%, but it is possible that careful observation of patients after treatment for signs of tetany would show that this is more frequent. We have observed epileptiform convulsions in four patients during disintoxication from soneryl. In each of these cases we were able to exclude personal or family history of epilepsy :

CASE 1.—(Described above.)

CASE 11.—(Described above.)

CASE 32 : *Single woman, aged 22. Melancholia.*—The heredity is unsound ; the mother has suffered from a melancholic attack, the patient's elder sister is a chronic psychoneurotic, while the youngest sister died from pulmonary tuberculosis.

The patient was of happy disposition until the death of her sister after a long and distressing illness. The psychosis began with insomnia and loss of appetite. She became more and more depressed and agitated, thinking and speaking of nothing but her own impending death. She made several doubtful attempts at suicide and then resolutely refused all food. At this stage she was admitted to the hospital as a voluntary patient. On admission she was noisily depressed, restless and resistive to attention, refusing all food and soon needing to be artificially fed. She was given a course of soneryl narcosis for seventeen days, during the whole of which time she had the maximal amount of sleep. Her temperature remained normal throughout the treatment and her pulse was good until the thirteenth day, when it dropped to 52. The following day it had slowed down to 50. Dosage was lessened, and the treatment was continued for four days longer. All this time she had taken food freely, her craving being for rice pudding. The first day after treatment she seemed dull and slightly confused. The second morning after treatment she had a major epileptic fit, followed by a temporary lucidity in which she was calm, clear and rational in conversation, but complained

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of an exceedingly severe headache over the vertex. It is noteworthy that this is the only time she has been known to complain of headache. Unfortunately she quickly relapsed into her former state of depression.

CASE 37 : *Single man, aged 25. Schizophrenia (criminal lunatic).*—He was admitted in December, 1935, and during the following six months was persistently noisy, truculent, interfering and destructive, with outbursts of violence. Delusions of persecution that his food was poisoned, and that his body was wasting away, alternated with feelings of unreality and vague ideas of reference. He exhibited marked dissociation of mood and thought, attributing his misfortunes to the fact that his sister was casting a spell over him. Hallucination of voices made him very noisy at night, so that he always needed one and sometimes two draughts of paraldehyde. He was given a course of soneryl narcosis during which he received gr. x *b.d.*, and slept most of the time. At times he talked incoherently, but he was not noisy. His habits were clean, and he ate readily throughout the treatment. Temperature and pulse remained normal the whole time. Three days after treatment he had a severe fit, following which he complained of headache lasting about an hour. This patient did not recover, but the following improvement has been observed : (1) Clean and looks after personal needs, (2) attempts to occupy himself, (3) will converse rationally and coherently, (4) sleeps without hypnotics, (5) far more amenable and co-operative, and (6) his hallucinations are becoming less intense.

Note.—Cases 1 and 11 received insulin throughout the period of narcosis. Cases 32 and 37 did not receive insulin. Furthermore, in Case 1 there was the additional factor of calcium loss during pregnancy and the puerperium.

SUMMARY OF COMPLICATIONS.

The various toxic phenomena which are known to accompany prolonged narcosis were looked for in the present series. Some have been described already. Others, such as troublesome pyrexia and lung affections, were absent. All the complications which occurred are enumerated as follows :

Carbohydrate metabolism.—Slight disturbances were seen in 75% of treatments. The commonest was a transient acetonuria, seen in 30 cases, but it was never severe, and invariably disappeared after the administration of insulin. Slight glycosuria was common and probably due to excessive intake of glucose. It was not considered important.

Cardio-vascular.—Minor disturbances occurred in 10% of treatments. The most important complication was marked bradycardia, which in one case reached a degree suggesting complete heart-block. In two other cases narcosis was discontinued on account of pulse-rates of 42, with a systolic blood-pressure below 100. Tachycardia was not observed except in Case 10, the patient who died from circulatory failure.

Gastro-intestinal.—Three patients vomited during the early stages of narcosis, but it was not necessary to interrupt the treatment, and it sufficed to reduce the dose of soneryl and to increase the insulin. Dysphagia, which has been described by most authors, was not encountered, and in the whole series only three patients refused food.

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Respiratory.—Nil.

Genito-urinary.—Slight disturbances occurred in 30% of cases. The commonest was a transient albuminuria, which disappeared again without trouble. Microscopical examination of deposits showed scanty red blood-cells in two cases, but there were no casts or other signs of renal damage. Three early cases were catheterized on account of retention associated with oliguria, but since then we have followed Parfitt's (12) suggestion of giving 100 oz. of nutrient fluid daily, and the complication has not recurred.

The skin.—Herpes labialis occurred in one patient during treatment. Profuse sweating occurred in one case, but there were no other skin complications.

The temperature.—Severe changes of temperature were not seen in this series. Slight pyrexia (99°) occurred during three treatments, and transient pyrexia after treatment was seen in three more cases.

Epilepsy.—Four cases are described above.

Psychic complications.—One case became hallucinated during treatment. Three melancholics became catatonic for periods during treatment.

Mortality.—The one patient with circulatory collapse after infusion would have died in any case, and it is unlikely that her condition was aggravated by narcosis.

Therapeutic Results.

The classification of results into Recovered, Relieved and Not Improved fails to differentiate that very interesting group of patients whose improvement, though marked, is only temporary. These cases in which temporary recovery is followed by relapse are most instructive. They indicate that narcosis alone is insufficient, and that where psychological readjustment is not effected there is little hope of permanent recovery. We have followed MacNiven's (10) scheme of tabulating our results in four groups :

(1) *Recovered.*—Rapid and complete restoration to health.

(2) *Relieved.*—Rapid and marked improvement, not however amounting to complete recovery. (*Note.*—Many of this group afterwards progressed to full recovery.)

(3) *Temporarily improved.*—Apparent recovery, followed within a few weeks by relapse.

(4) *Not improved.*—Self-explanatory.

Results in Schizophrenia.

Type.	Recovered.	Relieved.	Temporarily improved.	Not improved.
Catatonic stupor	0	2	4	6
Autistic	1	2	0	2
Excited	0	3	0	0
	1	7	4	8

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The results in some cases of schizophrenia are encouraging in others disappointing. In view of the fact that 12 cases treated were very chronic and supposedly incurable, the percentage recovery is inevitably low. However, of the 8 earlier cases treated, 1 recovered and 7 were relieved. The quickest response to treatment was seen in the autistic cases, of which the following is typical:

CASE 38: *Single man, aged* 35.—On admission he was persistently lethargic and exhibited marked dulling of affect. He seldom spoke, but when questioned, volunteered vague hypochondriacal delusions. He would do nothing for himself, and had to be persistently encouraged before he would eat. He was in fairly good physical health, except for slight cardiac enlargement with a mitral systolic murmur conducted out to the axilla. He was given a course of soneryl for 9 days. He immediately became brighter and more alert, with a new interest in his personal appearance. He was considered to be convalescent, and was occupied in the handicraft department.

Results of Manic-Depressive Psychosis.

Type.	Recovered.	Relieved.	Temporary improvement.	Not improved.
Mania	1	5	0	2
Melancholia	1	5	4	0
Involutional melancholia	0	0	1	1
	2	10	5	3
		20		

The fact that our recovery-rate in this group is somewhat lower than that of other writers is counterbalanced by the considerably lower incidence of toxic complications.

The following is a typical result on a case of mania:

CASE 12: *Married woman, aged* 31.—She was admitted with a history of 6 weeks of increasing insomnia, irritability and excitement. Her condition was one of constant over-activity. She sang and shouted incoherently, would not stay in bed, and was persistently restless, interfering and threatening. She was given 3 days' light narcosis, during which she was most resistive and had to be tube-fed. Then narcosis was pushed very deep for 5 days, during which she obtained the maximum amount of sleep. Her pulse dropped to 46, but her general condition remained satisfactory. Five days after treatment she changed suddenly and became amenable, friendly and co-operative. A few days later she was moved to an open ward, where she made a good recovery.

The next case illustrates the response to treatment in agitated melancholia:

CASE 13: *Married woman, æt.* 51.—On admission she was persistently depressed and lachrymose, with much agitation and constant requests for help, though she said no one could help her, and that her misery was a punishment brought on her family. She stated that scandal and dishonour

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had been brought to her house (this was untrue). She was excited, restless and incoherent. She was given narcosis for a week. Following this she became mildly overactive for a few days, but did not develop a manic phase. Her recovery was rapid, complete and uneventful.

Conclusion.

Soneryl narcosis produces marked improvement and sometimes recovery in many intractable psychoses.

It may be compared with somnifaine narcosis, and even if the recovery-rate is rather lower at least the toxic complications are less common.

Narcosis induced by the method of continuous intravenous infusion, can be applied to those cases described as definitely unsuitable for somnifaine therapy, viz., the acute toxic psychoses and the exhaustions from mania and melancholia.

I am indebted to Dr. P. W. Bedford, Medical Superintendent of the Dorset Mental Hospital, for his interest and encouragement in this work. My thanks are due also to Dr. G. T. James for his notes on the male cases and for his assistance in many of the treatments.

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THE EXCRETION OF SODIUM BARBITONE IN PATIENTS SUFFERING FROM MENTAL ILLNESS.

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THE subject of the fate of sodium barbitone in the body appears still to be a valid subject for discussion; various workers have investigated the excretion of the drug in the urine of normal persons and persons suffering from bodily—not mental—illness.

Reiche (1) and Reinert (2) from analyses of the urine following ingestion of barbitone found that the drug was excreted very slowly: 8% appeared in the urine in the first 12 hours following administration 20%, in 24 hours, 36% in 48 hours, and traces of the drug appeared for as long as nine days after a single dose.

Frethwurst, Halberkann and Reiche (3) were able to recover a total of 75% of a given dose of sodium barbitone from the urine. Of the remaining 25% the fate was unknown. The question of excretion is one of practical importance, since Willcox (4) has shown that the repeated use of many barbiturate drugs is attended by considerable danger, and may be very harmful. Mutch (5) emphasizes the danger of hang-over and cumulation of these drugs.

On the other hand, in the opinion of Craig (6), Gillespie and others, there is little danger from the prolonged administration of these drugs, and they consider them to be the most useful in psychiatric practice.

The present investigation has the following purposes:

(1) To. ascertain a reliable method of measuring the excretion of sodium barbitone in the urine.

(2) To determine the rate of excretion of sodium barbitone in a number of patients.

(3) To ascertain whether the rate of excretion was in any way related to the efficiency of the renal functions.

(4) To ascertain whether any relationship existed between the type of mental illness and the rate of sodium barbitone excretion.

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Method of Investigation.

It was decided to investigate the excretion of sodium barbitone in the following way: 24 male patients suffering from various well-marked forms of mental illness were selected; the ages varied between 25 and 70. These patients were in good bodily health, clean in their habits, up from bed, and for the most part engaged in routine hospital tasks. For 7 days before the procedure about to be described they were on an ordinary diet, and were given no medicines apart from the routine aperients. Each was then given a dose of 648 mgrm. of sodium barbitone (10 gr.). The urine for the next 24 hours was collected and the amount of sodium barbitone estimated. At the same time the urea clearance was also estimated to get an indication of the degree of renal efficiency.

To determine the amount of sodium barbitone present in the 24-hour specimen the method of Itallie and Steenhauer, modified by Straub and Mihalovitis (7) was employed; this was selected on account of its relative simplicity and accuracy, and is as follows:

400 c.c. of the 24-hour specimen are taken and placed in a 2-litre Erlenmeyer flask and heated to 80°–90° C. 25 c.c. of 10% sulphuric acid are added. Then as much saturated potassium permanganate solution is added, so that, after shaking up the abundant brown sediment deposited on the sides of the flask, the back-flowing liquid remains violet for 10 minutes.

Drops of 30% strong perhydrol (Merck's) are added to the violet-coloured solution containing the deposit until a crystal-clear solution is obtained. To the solution at 70°–80° C. is added 0·4 grm. of medicinal charcoal (Merck).

It is then shaken for a few minutes, filtered, and the residue on the filter-paper thoroughly washed with 4 volumes of 15 c.c. of hot, distilled water. The colourless, crystal-clear liquid is then shaken in a separating funnel with 5 volumes of 50 c.c. of ethyl acetate. The extract is filtered, and the filter-paper washed with ethyl acetate. This is then allowed to evaporate in a beaker at a temperature of 50°–60° C.; the barbitone is deposited as white, needle-shaped crystals, and should have a melting-point of 189°–191° C.

Practical Difficulties and Modifications.

The originators of the method worked with urine to which a known amount of barbitone was added, and state that the barbitone is deposited in snow-white crystals. In the case of ingested barbitone, however, we find that this is not so, and the melting-point is actually lowered. The following additional procedure was necessary in order to obtain a pure specimen giving a reasonably

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accurate melting-point. Roche Lynch emphasizes the fact that the actual melting-point in all these estimations is several degrees lower than the theoretical melting-point.

After the ethyl acetate extract has evaporated, a brownish deposit containing crystals is obtained. The melting-point at this period is between 150° and 160° C. The brown deposit is dissolved in a little hot distilled water, acidified with acetic acid. A slight amount of charcoal is added, and it is allowed to stand for a short time and is then filtered, the filter-paper being washed with a little hot distilled water. The filtrate is then treated four times with 10 c.c. of ether and the ether extract filtered into a small beaker of known weight. By means of a rotary pump a small current of air is directed on to the surface of the ether, which is thus evaporated. When nearly all the ether has evaporated the mixture in the vessel freezes ; it is then placed in a bath at 50° C., and the evaporation is allowed to proceed to completion and dryness. This is allowed to proceed overnight, although after a very short time white crystals can be seen on the bottom and sides of the vessel. When complete the weight is taken, and the melting-point determined.

In some cases the crystals are not quite pure, so the procedure is repeated, and pure crystals are almost always obtained. During this process of evaporation it was found that approximately 2 mgrm. of barbiturate was lost by evaporation from the 250 c.c. of ethyl acetate solution ; this loss was fairly constant, irrespective of the total amount of sodium barbitone in solution, so that the inevitable error due to this factor is of greater import in the cases with lower total yield. Roche Lynch emphasizes the fact that the melting-point is always a few degrees below that of the pure product, and Straub and Mihalovitis point out that when using measured amounts of barbitone more was obtained than was put into the urine by reason of impurities.

The tests employed to identify the crystals as those of sodium barbitone were :

(1) Melting-point.

(2) Millon's test.

Dissolve some of the crystals in a minimum amount of water. Take a drop of this solution and acidify with a fraction of a drop of dilute nitric acid, and add a drop of Millon's reagent. A white precipitate denotes the presence of barbiturates.

(3) Cobalt nitrate test.

Solution A : Cobalt nitrate 1 grm. ; absolute alcohol to 100 c.c.
Solution B : Potassium hydrate 1 grm. ; absolute alcohol to 100 c.c.

To a few of the crystals add 0·5 c.c. of a mixture of chloroform 3 parts and absolute alcohol 1 part. Add a few drops of solution A, and then cautiously add solution B drop by drop. The presence of barbiturates is shown by an immediate blue colour discharged by excess of solution B.

(4) Microscopic examination.

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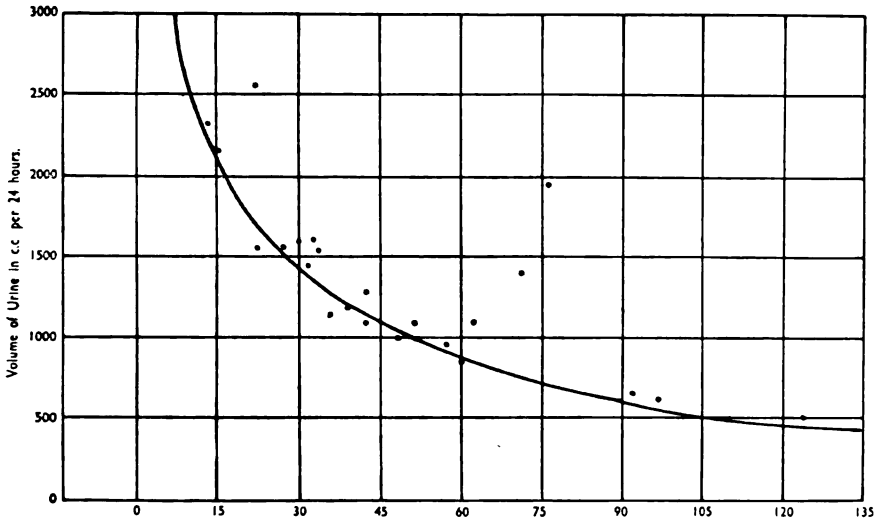
The other method available was that quoted by Roche Lynch, stated to be a quick method for use in cases where the barbiturate is excreted in fair quantities, but this did not appear to be so satisfactory in dealing with small amounts.

Another source of difficulty was encountered with regard to obtaining accurate 24-hour specimens of urine. All the patients selected were males who were clean in their habits, but several could not be relied upon for collecting a 24-hour specimen. As far as possible this was supervised by the staff. It

CHART I.



Titratable acid and fixed ammonia expressed in terms of N/10 NaOH per 100 c.cm. of urine.

became necessary during the course of the investigation to keep the patients in bed during the collection of the specimen. In addition, in cases where the accuracy of the 24-hour volume was at all in doubt, an attempt was made to check up the volume by means of the following method: According to work done by Palmer and Henderson (8) the total titratable acid plus fixed ammonia in the urine expressed in terms of N/10 NaOH% bears a relationship to the total volume of urine within fairly close limits. This enables a reasonably close estimate to be made of the total output of urine in 24 hours. The relationship indicated by Palmer and Henderson's work is not strictly rectilinear, but can be expressed in the form of a graph, and this has been done by Dr. A. F. Sladden, who has used it for many years.

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This chart, in our own and Dr. Sladden's experience, corresponds in most cases closely with the observed 24-hour total. Utilizing this principle the actual volumes recorded could be checked by reference to the "theoretical" volume as shown on the graph. In any case where the volume of the 24-hour specimen was in doubt, the titratable acid plus fixed ammonia was estimated and compared with the graph. The actual volume could then be checked by comparison with the volume obtained by means of the graph.

The urea clearance in each case was estimated by Van Slyke's method, modified by estimating the urine output per minute on the basis of the actual 24-hour output, and the renal efficiency expressed in terms of percentage of average normal function. In good renal health this percentage may range from 70 to 120. In all cases the urine was thoroughly investigated by routine methods.

In each case all the above investigations were repeated after an interval, and in some cases they were carried out three and even four times to ascertain how far there was uniformity of results in each individual case. In all 24 cases were investigated.

Results.

These are best expressed in the form of the following tables:

TABLE I.—*Showing the Amount of Sodium Barbitone Excreted in the First 24 Hours after the Administration of a Dose of 648 mgrm. in the Series.*

Case No.	Total volume 24-hour urine. c.c.	Sodium barbitone recovered from— 400 c.c. urine (mgrm.).	24-hour urine (mgrm.).	Percentage of intake recovered.	Melting-point (°C.).
1	625	62	97·0	15·0	180
	700	88	154·0	23·7	180
	625	40	62·5	9·6	179
2	650	43	69·875	10·78	176
	1,600	25	100·0	15·4	178
3	1,200	36	108·0	17·0	180
	1,100	52	143·0	22·0	174
4	800	11	22·0	3·4	..
	1,300	18	58·5	9·0	176
	700	25	43·75	6·7	184
5	2,350	21	123·0	19·0	182
	1,900	32	152·0	23·4	178
	1,000	25	62·5	9·6	187
	2,150	23	123·625	19·1	182

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TABLE I—*continued.*

Case No.	Total volume 24-hour urine c.c.	Sodium barbitone recovered from— 400 c.c. urine (mgrm.).	24-hour urine (mgrm.).	Percentage of intake recovered.	Melting-point (°C.).
6	720	15	27·0	4·1	176
	1,550	7	27·125	4·2	..
7	1,275	22	70·136	10·8	178
	800	41	71·75	11·0	183
8	1,200	29	87·0	13·4	183
	2,200	12	66·0	10·2	182
9	1,000	41	102·5	15·8	177
	1,300	22	71·75	11·0	185
10	1,150	18	51·25	7·9	183
	900	35	78·75	12·0	186
11	600	18	27·0	4·16	178
	400	18	20·6	3·16	180
12	1,050	22	57·75	8·9	176
	1,000	15	37·5	5·78	..
13	750	34	63·75	9·8	176
	1,450	24	87·0	13·7	176
14	2,050	10	61·5	9·49	180
	1,350	22	73·26	11·3	182
15	900	28	63·0	9·7	180
	600	40	60·0	9·2	185
16	1,000	27	67·5	10·4	183
17	1,200	28	84·0	13·0	180
	950	37	87·875	13·5	183
18	1,550	33	127·85	19·7	183
	500	66	82·5	14·0	186
19	1,300	13	42·5	6·5	182
	1,950	9	43·875	6·6	..
20	1,150	28	89·5	12·56	180
	2,550	12	76·5	11·8	..
21	1,600	21	84·0	12·5	183
	1,100	25	68·75	10·6	184
22	850	8	17·0	2·62	..
	1,050	11	28·875	4·45	182
23	650	32	52·0	8·0	183
	2,300	18	103·5	16·0	182
24	1,100	23	63·25	9·7	183
	1,550	18	47·25	10·1	184

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TABLE II.—*To show the Relationship between the Excretion of Sodium Barbitone and the Degree of Renal Efficiency Expressed as a Percentage of Average Normal Function* ($N = 70$–120).

Case No.	Urine urea mgrm. per 100 c.c. urine.	Urea clearance % of average normal function.	Barbitone mgrm. per 100 c.c. urine.	$\frac{\text{Urea}}{\text{Barbitone}}$ ratio.
1	2,660	82	15·5	177 : 1
	1,656	53	22·0	75 : 1
	1,500	59	10·0	150 : 1
2	3,000	108	10·75	279 : 1
	1,560	106	6·25	249 : 1
3	1,350	54	9·0	150 : 1
	1,746	59	13·0	134 : 1
4	2,376	66	2·75	864 : 1
	1,128	68	4·5	251 : 1
	1,476	62	6·25	236 : 1
5	1,218	77	5·25	232 : 1
	1,182	69	8·0	148 : 1
	1,944	98	15·625	310 : 1
	1,170	87·5	5·75	203 : 1
6	2,124	50	3·75	566 : 1
	1,998	65	1·75	1,141 : 1
7	1,188	53	5·5	216 : 1
	1,386	55	10·25	135 : 1
8	2,380	82	7·25	328 : 1
	810	51	3·0	270 : 1
9	2,838	98	10·25	276 : 1
	2,196	94	5·5	399 : 1
10	2,226	78	4·5	494 : 1
	2,886	96	8·75	329 : 1
11	1,758	63	4·5	390 : 1
	2,124	46	5·1	410 : 1
12	1,700	63	5·5	309 : 1
	1,752	54	3·75	467 : 1
13	2,290	97	8·5	269 : 1
	1,260	77	6·0	210 : 1
14	750	29	3·0	250 : 1
	1,836	51	5·5	334 : 1
15	2,394	88	7·0	342 : 1
	3,510	102	10·0	351 : 1
16	1,596	75	6·75	236 : 1

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TABLE II—*continued.*

Case No.	Urine urea mgrm. per 100 c.c. urine.	Urea clearance % of average normal function.	Barbitone mgrm. per 100 c.c. urine.	$\frac{\text{Urea}}{\text{Barbitone}}$ ratio.
17	1,242	92	7·0	177 : 1
	2,046	88	9·75	209 : 1
18	1,590	101	8·25	192 : 1
	3,402	90	16·5	206 : 1
19	1,836	120	3·25	565 : 1
	1,086	96	2·25	482 : 1
20	1,721	100	7·0	246 : 1
	786	80·5	3·0	262 : 1
21	1,194	63	5·25	227 : 1
	1,344	84	6·25	215 : 1
22	2,046	106	2·0	1,023 : 1
	1,974	82	2·78	710 : 1
23	3,070	97·6	8·0	383 : 1
	570	59	4·5	127 : 1
24	1,614	82	5·75	280 : 1
	1,196	76	4·25	281 : 1

TABLE III.—*Showing the Relationship between* (a) *the Type of Mental Illness ;* (b) *the State of Renal Function ;* (c) *the Excretion of Sodium Barbitone.*

Case No.	Type of mental illness.	Urea clearance % of average normal function.	Barbitone % of intake recovered.
1	Primary dementia	82·0	15·0
		53·0	23·7
		59·0	9·6
2	,, ,,	108·0	10·78
		106·0	15·4
7	,, ,,	53·0	10·8
		55·0	11·0
8	,, ,,	82·0	13·4
		51·2	20·3
10	,, ,,	78·7	7·9
		96·0	12·0
13	,, ,,	97·0	9·8
		77·0	13·7

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TABLE III—*continued.*

Case No.	Type of mental illness.	Urea clearance % of average normal function.	Barbitone % of intake recovered.
17	Primary dementia	92·0	13·0
		88·0	13·5
18	,, ,,	101·0	19·7
		90·4	14·0
20	,, ,,	100·0	12·56
		80·5	11·8
11	Mental deficiency	63·0	4·16
		46·0	3·16
22	,, ,,	106·0	2·62
		82·0	4·45
21	,, ,,	63·0	12·5
		84·0	10·6
19	,, ,,	120·0	6·5
		96·0	9·0
15	Manic-depressive insanity	88·5	9·7
		102·0	9·2
3	,, ,,	54·0	17·0
		59·0	22·0
12	,, ,,	62·9	8·9
		54·4	5·78
9	Insanity of gross brain lesion	98·0	15·8
		94·0	11·0
5	,, ,, ,,	77·0	19·0
		79·0	9·6
		87·5	19·1
6	Delusional insanity	50·0	4·1
		65·0	4·2
16	General paralysis	75·0	10·4
4	Epilepsy	66·0	3·4
		68·0	9·0
		62·0	6·7
23	,,	97·6	8·0
		59·0	16·0
24	,,	82·0	9·7
		76·0	10·1
14	,,	29·5	9·49
		51·15	11·3

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TABLE IV.—*Showing the Influence of the Re-purification Method on the Yield of Sodium Barbitone Obtained in 6 Cases taken at Random.*

Case No.	Weight of barbitone and impurities recovered from 24-hour urine (mgrm.).	Expressed as percentage of intake	Weight of pure barbitone from 24-hour urine (mgrm.).	Expressed as percentage of intake.
24	143	22·0	63	9·7
23	82	14·0	52	8·0
22	48	7·6	17	2·6
21	160	24·0	84	12·5
8	132	20·0	66	10·2
18	182	28·0	127	19·7

DISCUSSION.

(1) Those cases which show definite renal impairment appear to have an impaired rate of sodium barbitone excretion as compared with other cases in which the renal efficiency is normal. The relationship between renal efficiency and barbitone excretion may conveniently be expressed as in Table II by means of the urea/barbitone ratio. In cases with normal renal function this would seem to vary between 150 : 1 and about 300 : 1.

At first sight the limits of variation appear to be very wide, but this apparently large margin of normality can be correlated with the limits of normality of tests of renal function. Figures between 70 and 120 are regarded as the normal limits of average renal function, and the difference between the upper and lower limits is only relatively little less than between those of the figures given for the urea/barbitone ratio.

In those cases in which the renal efficiency is definitely impaired (Cases 4, 6 and 11) the ratio rises to much higher figures. For example, in Case 4 the average of three results gives a urea/barbitone ratio of 411 : 1; in Case 6 803 : 1; in Case 11 400 : 1. It would appear, therefore, that a failure in the efficiency of the kidneys extends to the barbiturate drugs more than to the urea, possibly owing to the larger size of the barbiturate molecule.

(2) Apart from the renal efficiency, the amount excreted appears to show a little variation in the different types of mental illness; e.g., in the series of cases of primary dementia with average normal renal function, the average percentage of sodium barbitone recovered was 13·67. In the case of manic-depressives it was 12·1. In cases of insanity with gross brain lesion it was 14·9. In cases of epilepsy it was 10·76. It would, however, be necessary to follow up the differences noted in this connection in a larger series of cases before any valid conclusions could be drawn.

(3) From the above figures it will be seen that notably in cases suffering

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from epilepsy, the percentage of sodium barbitone excreted is less than that which would be expected from the results of previous workers on the rate of sodium barbitone excretion. The question arises whether this small percentage is in some way due to the influence of the disease, viz., epilepsy, on the metabolism, or whether it is due to the method of re-purification adopted in these experiments, producing specimens of sodium barbitone in a state of greater purity and consequently less bulk.

It would appear that both factors are operative. The re-purification method was adopted in all the cases, and still taking epilepsy as our example, the amount recovered from these cases is smaller than in cases of other types of mental illness, the renal efficiency being in all cases within normal limits. A striking example of this occurred in Case 23. At one estimation we recovered 16% of the 10-gr. dose, and on this occasion he had been free from fits for more than a week previously. On the previous occasion he had had several major fits and was in a very confused state, and here, using the identical technique, only 8% of the dose was recovered.

But, quite apart from the type of mental illness present, practically all the figures obtained in these experiments are less than the 20% of intake referred to by other workers. Straub and Mihalovitis point out that, after using the original method of Itallie and Steenhauer the impurities obtained amount to as much as 50 mgrm. from 400 c.c. of urine, and they appear to weigh this impurity as barbitone in their results. Consequently they frequently obtained more barbitone from the urine than they added.

Even after using the modification of Straub and Mihalovitis we found impurities still to be present. If these were weighed as barbitone our figures for percentage of intake recovered reach and in some cases exceed the quoted 20%. These figures are shown in Table IV, which compares the amount of barbitone and impurities with the amount of pure barbitone obtained after re-purification in six of the cases selected at random. It will be seen that in many cases the amount of impurity exceeds 50% of the total.

Summary.

(1) A method of estimating the excretion of sodium barbitone in the urine is described, with modifications and suggestions for overcoming inherent difficulties.

(2) The results in 24 cases are given, and are correlated with the renal efficiency in the form of urea/barbitone ratio.

(3) The relationship between the type of mental illness and the sodium barbitone excretion is discussed.

(4) The amount of impurity to be expected is discussed, and a technique for re-purification described and the results given.

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We wish to express our indebtedness to Dr. N. Moulson, Medical Superintendent of Cefn Coed Hospital, for his help and permission to publish this paper, and to Dr. A. F. Sladden, Visiting Pathologist, for his constant and valuable help.

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SOME OBSERVATIONS ON ENDOCRINES IN THE EMOTIONAL PSYCHOSES.*

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IT has long been recognized that disturbances of the thyroid, such as cretinism, myxœdema and some cases of Graves's disease, can give rise to mental symptoms. Of recent years, however, other endocrinopathies have been investigated from the psychological standpoint, and in nearly all cases evidence of some abnormality has been found. For example, Werner (1) examined 53 castrates, 48 cases of involutional melancholia, and 96 women of menopausal age, and found marked similarity between the mental symptoms occurring at the menopause and those in involutional melancholia, and presumed that they were both due to the same cause—ovarian dysfunction.

McCartney (2) examined 20 Chinese eunuchs and 3 Skoptsi (members of a Russian religious sect practising castration in the first decade of life), and found that they showed typical schizoid symptoms, such as apathy, emotional dullness, loss of initiative and undue tendency to introspection, without any intellectual impairment. He also found evidence of gonadal dysfunction on examination of a large number of male and female schizophrenics. Engelbach (3) found evidence of hypersexuality in the early stages of many of his cases of hyperpituitarism and suprarenalism, which is later replaced by loss of libido. Power (4) has emphasized Cannon's findings on the part played by the suprarenals in the emotions of fear and rage, and suggests that probably all the other emotions are similarly implemented by endocrine secretions, and that the latter may therefore be assumed to play a large part in the purely emotional psychoses, i.e., manic-depressive insanity and schizophrenia.

Finally there is the classical work of Mott (5) on the regressive changes occurring in the testes and ovaries in schizophrenia.

ENDOCRINOLOGY.

The only really definite proof of existing endocrine dysfunction would be the demonstration of an excess or diminution of the particular hormones in the blood, but at present, with the exception of the Ascheim-Zondek reaction as a test for the presence of the gonadotropic principles of the anterior pituitary in the urine and blood in pregnancy, this is not yet possible. In the meantime diagnosis must be

* Read at the meeting of the Northern and Midland Division October 29, 1936.

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made by the consideration of morphological deviations of the body and the laboratory investigation of metabolic changes. Unfortunately the findings resulting from the latter are too variable to serve as diagnostic features by themselves, with the possible exception of the estimation of the basal metabolism in thyroid disorders, so that at present one must fall back on morphology as the most useful indication of endocrine functioning. In the case of the gonads a history of disordered function is helpful, but it is possible for states of definite hypogonadism to exist which are not severe enough to interfere with reproductive functions. No doubt when completely reliable and standardized extracts of the various glands are available it will be possible to confirm any diagnosis of endocrine disorder by noting the response to treatment, just as many authorities now consider that a beneficial response to iodine therapy substantiates a diagnosis of hyperthyroidism.

The endocrines are among the most important regulators of both the pre- and post-natal development of the human form, and therefore abnormalities of the latter are significant of endocrinopathy, provided those features are selected which are known to be solely due to endocrine influence. These morphological deviations can be ascertained by two methods of approach—inspection and anthropometry. By inspection one can note the amount and relative distribution of the body-fat and hair, the texture of the skin, and the degree of muscular development, and by anthropometry one can determine the proportionate degrees of skeletal development. In this connection it may not be out of place to remind the reader of some of the functions of the endocrines as so far ascertained.

The anterior lobe of the pituitary elaborates hormones which promote body growth and the maturation of the sex-glands, also controlling fat metabolism and the activities of the thyroid, parathyroids, pancreas and adrenal cortex. The posterior lobe is thought to elaborate pressor and oxytocic principles. Of all these, the effect of the growth hormone on the development of the bones is the most definite and reliable indication of pituitary hyper- or hypo-function. Hyperpituitarism occurring before puberty produces overgrowth of both long and flat bones, the increase in the case of the former being both in length and breadth. Should the onset occur after the age of epiphysial union, the enlargement is confined to the flat bones and to increase in the width of the long bones; i.e., the changes found in acromegaly. Hypoactivity of the growth hormone is only apparent in cases in which the onset occurs in infancy, resulting in a dwarf with child-like proportions, i.e., the Lorain-Levi syndrome. Hyperactivity of the gonadotropic hormone is not easy to distinguish owing to the uncertainty, at present, as to how far the symptoms may be attributed to the pituitary or to the suprarenals. Hypoactivity of this hormone produces the well-known Fröhlich's syndrome, in which genital hypoplasia is associated with marked obesity, involving mainly the regions of the breasts, mons, pelvic and shoulder girdles (Figs. 1 and 2). This condition, which is most common in adolescence, may also arise in adult life, and then the genital symptoms accompanying the obesity are purely functional and without anatomical change. Disorders of the remaining pituitary hormones do not produce any easily recognized morphological signs and were not investigated.

The internal secretion of the testis is responsible for the development of the secondary sex characteristics and for the epiphysial union of the long bones.

Deficiency of this secretion causes slightly different symptoms according to whether the time of onset is before or after adolescence. If before, the lack of normal stimulus to epiphysial union results in the long bones continuing to grow beyond their normal time, ultimately producing arms and legs which are out of all proportion to the total stature. This can be seen in Fig. 3, which shows two males of equal height, and each with the anterior superior iliac spines marked with ink, in order to show the comparative length of the legs. It is obvious that the man on the left is equal in height to that on the right, merely because of greater length of leg. Actually the measurements from the upper margin of the symphysis pubis to the soles of the feet should be almost equal to that from the vertex to the upper margin of the symphysis pubis, as it is in the man on the right, where epiphysial union had occurred at the correct time in response to endocrine stimulus

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from the testes. The first man, or youth, was a case of moral imbecility without intellectual impairment. He presented the physical signs of delayed puberty, viz., overgrowth of arms and legs, a childish and narrow face, and although seventeen years of age still had a treble voice and had never had to shave.

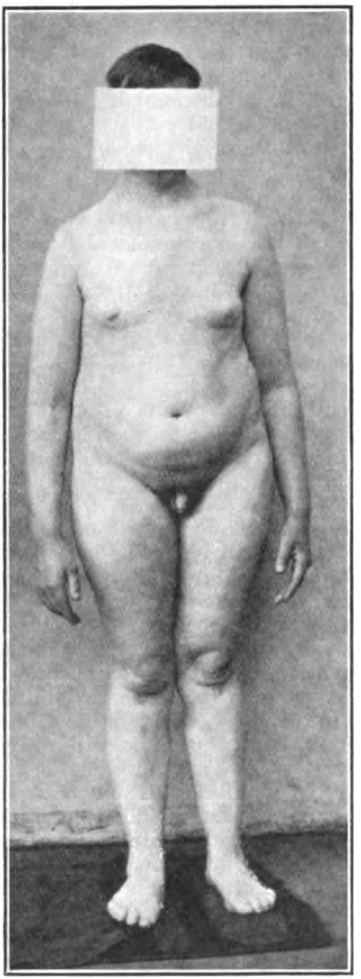

FIG. 1.

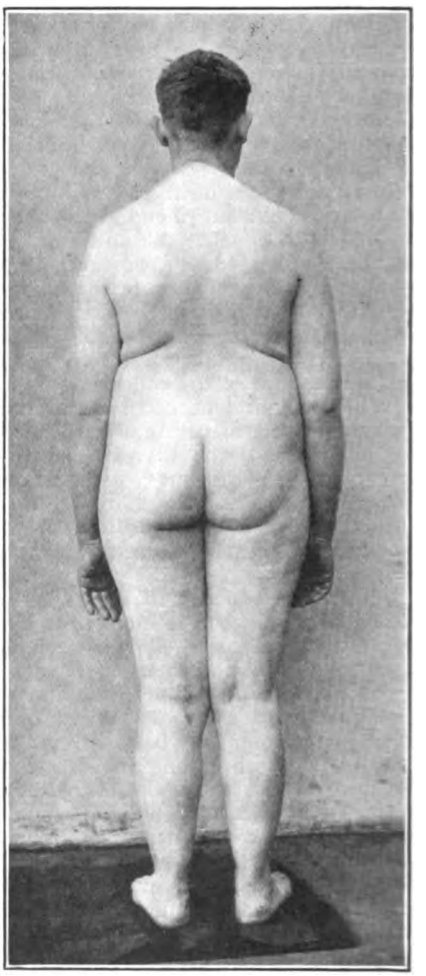

FIG. 2.

Typical obesity in hypopituitarism.

There was no axillary hair and the pubic growth was very scanty. The genitalia were those of a boy of about twelve years of age.

This disproportion between the upper and lower measurements is also dependent on normal functioning of the pituitary growth hormone, and if the latter is deficient it will not occur, the individual remaining a dwarf of more or less symmetrical proportions. But as the stimulus for the maturation of the testes comes from the

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pituitary, any marked increase of the lower measurement over the upper always indicates hypogonadism, no matter whether its origin is primarily in the gonads themselves or in the pituitary. Hypogonadism arising after the period of active growth is over will not produce any modification of the skeleton.

Precocious development of the testicular hormone leads to premature closure

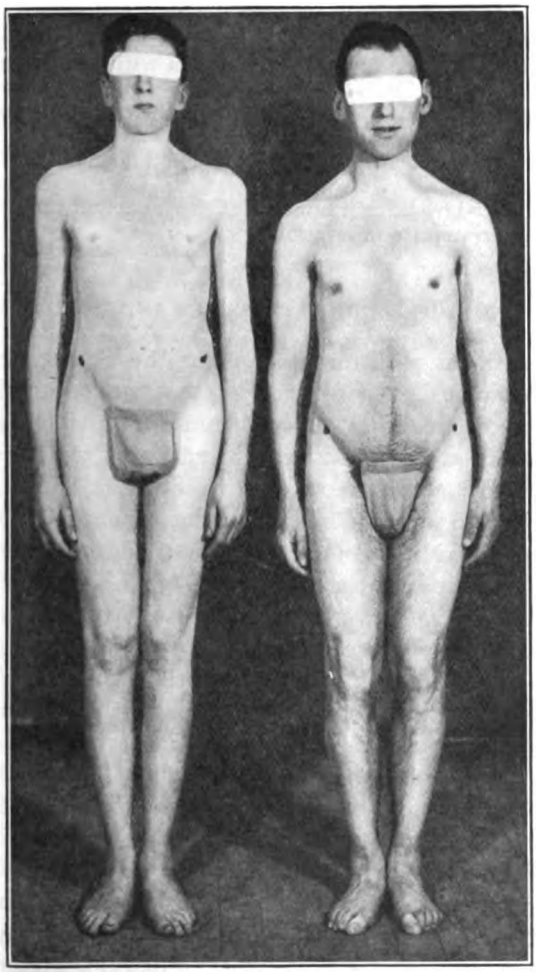

FIG. 3.

of the long bone epiphysis, so that hypergonadism occurring during the growth period produces the opposite effect, the upper measurement exceeding the lower, provided the pituitary growth hormone is unimpaired. The gonads also influence the development of the hair, fat, skin and muscle. Engelbach (3) describes the typical hypogonad as having well-developed primary hair (scalp and eyebrows), but poorly developed secondary hair (face, axilla, pubis, body, limbs), poor musculature,

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smooth-textured skin, narrowness of the lower part of the face, and a tendency to leanness, except during the later decades, when there may be a terminal obesity.

Hypergonadism, whether arising primarily, or as a result of suprarenal or pituitary abnormality, is associated with the opposite characteristics, viz., excessive development of the secondary hair and body musculature. This condition, which usually does not call for treatment unless occurring in females or children, is not necessarily entirely due to the gonads, but may be caused by some dysfunction of either the suprarenal cortex or the pituitary, or both combined. The function of the suprarenal cortex is thought to be that of a stimulant to the gonads, largely because of the association of suprarenal tumours with hirsutism and virilism in women, but the recent discovery that in some cases these changes can be caused by pituitary lesions has rather complicated the theory. Therefore any cases in the present investigation showing symptoms which are the direct opposite of hypogonadism have been labelled "hypergonadism", and no attempt made to implicate any particular gland.

The effects produced by the hormones of the thyroid and the suprarenal medulla are too well known to require reiteration.

Examination of Manic-depressives and Schizophrenics.

Two hundred male patients resident in the South Yorkshire Mental Hospital were examined, 100 of each of the two psychotic groups. The investigation was confined to males, because physiological alterations of endocrine balance do not occur so frequently in this sex as in women. The methods of examination used were: (1) inspection, (2) anthropometry.

On inspection note was made of the extent of muscular development, the general texture of the skin, the primary and secondary hair development, and the degree and distribution of the body-fat. Degrees of muscular development were grouped as (*a*) poor, (*b*) average, (*c*) increased; types of hair development as (*a*) good primary, but poor secondary hair, (*b*) average primary and secondary hair, (*c*) increased secondary hair; types of skin as (*a*) smooth, (*b*) average. These standards were arbitrary ones, formed after the examination of a large number of individuals, but since all cases were inspected by the same observer, the results are comparable. It was found that fat distribution did not lend itself very well to classification into groups, as this feature varies so much according to the state of the psychosis at the time of examination, so that only those cases were noted where the amount was very excessive and not to be accounted for by nutritional causes. The measurements taken were (1) stature (standing), (2) distance from the upper margin of the symphysis pubis to the soles of the feet (standing), (3) bizygomatic width (maximum width between the zygomatic arches), (4) bigonial width (distance between the angles of the mandible). No. 2 will, in future, be referred to as the lower measurement, from which the upper measurement may be obtained by subtracting it from the total stature. By dividing (4) by (3) and multiplying by 100 a figure, the bigonial index, is obtained, and is useful for representing statistically the shape of the face in a large number of people. Ordinary standard anthropometric apparatus was used for this part of the work. The results obtained were treated by

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standard statistical methods, i.e., by the use of "means" to express the characteristic measurements of each group, the standard error as a test of significance, and the χ^2 distribution as a means of testing the agreement between observation and hypothesis (6).

RESULTS.

TABLE I.—*Measurements.*

	Means and standard deviations.		Difference between means.	Standard errors.
	Manic depressives.	Schizophrenics.		
Stature	167·8 cm. σ 7·0	167·5 cm. σ 6·9	0·3 cm.	±0·97
Lower measurement minus upper	+2·0 ,, σ 5	+3·4 ,, σ 4·5	1·4 ,,	±0·69
Bigonial index	17·28 ,, σ 0·89	16·98 ,, σ 0·89	0·3 ,,	±0·12

It will be seen from the above table that although there is no significant difference between the mean statures of the two groups, there are small differences between the mean bigonial indices and between the means of the figures obtained by subtracting the upper from the lower measurement in each group. Since in each of these two latter cases the differences are twice the value of their respective standard errors, they may be considered to be significant. It was found, therefore, that the schizophrenics, as a class, had longer legs in proportion to their stature, and a more "inverted-egg-shaped" face, than the manic-depressives, both of which factors suggest a relative hypogonadism.

TABLE II.—*Muscular Development.*

	Poor.	Average.	Increased.		
Manic-depressives	20	57	23	=	100
Schizophrenics	34	41	25	=	100

$\chi^2 = 14{\cdot}6 \quad P. = 0{\cdot}01$

It will be seen from Table II that the two psychotic groups are dissimilarly distributed in respect to muscular development, and since the value of P. is below 0·05 this dissimilarity is not due to chance selection, but to the fact that they represent different anatomical groups. The greatest differences are between those of poor and average muscular development, the schizophrenics having a larger number of the former and a lesser number of the latter, from which one may conclude that schizophrenics as a class are poorer in muscular development than manic-depressives.

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TABLE III.—*Hair Development.*

	Primary good, secondary poor.	Average primary and secondary.	Increased secondary.		
Manic-depressives	9	20	71	=	100
Schizophrenics	21	25	54	=	100

$\chi^2 = 21{\cdot}3$ P. = 0·01

It will be noticed that there is a difference in the distribution of types of hair distribution, and again this is not to be explained by chance selection, the value of P. being below 0·05 in this table also. The two groups deviate in opposite directions, the schizophrenics tending to have good primary but poor secondary hair, whereas the manic-depressives tend to excessive secondary hair development.

TABLE IV.—*Texture of Skin.*

	Smooth.	Average.		
Manic-depressives	17	83	=	100
Schizophrenics	25	75	=	100

$\chi^2 = 4{\cdot}5$ P. = 0·03

This table shows that the distribution of the skin types also differs in the two classes (although the degree of difference is not so marked as in the case of the muscular and hair types), the soft-textured skin, typical of hypogonadism, being more frequently found amongst schizophrenics than manic-depressives.

All these findings, when pieced together, indicate that when schizophrenics are compared, as a class, with manic-depressives, they present morphological evidence of relative hypogonadism, being characteristically poor in muscular development and secondary hair, having a softer textured skin and a disproportionate growth of the long bones. The latter feature also indicates that the onset of the hypogonadism occurred during the active growth period. It is interesting to note how closely these features coincide with Kretschmer's leptosome type, which is said to be most frequent amongst schizophrenics.

Fig. 4 shows two selected patients, left a schizophrenic, and right a manic-depressive, each presenting graphically the characteristics of their class as found by this investigation.

As previously mentioned, fat distribution was only noted when excessive in amount, and that was in 26 manic-depressives and 6 schizophrenics. In all cases it was of the pituitary type of distribution, and in the case of the schizophrenics was associated with evidence of hypogonadism, whereas all the

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manic-depressive cases showed normal secondary sex characteristics, the obesity occurring late in life, as also did the onset of their psychoses in three-quarters of the cases. This latter fact may possibly mean that involutional

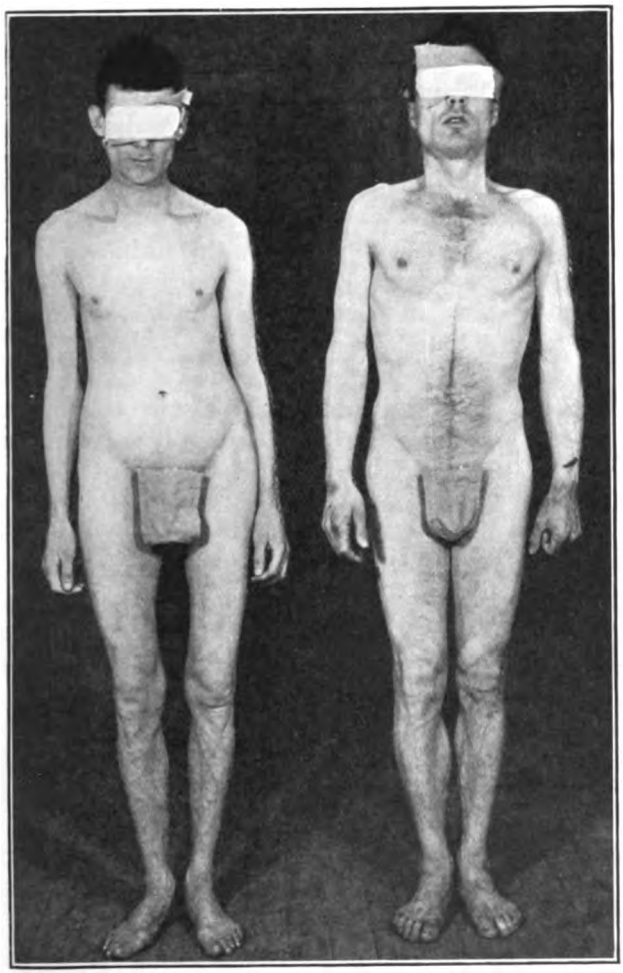

FIG. 4.

manic-depressive insanity has an endocrine basis. Here again there is some similarity between the endocrine morphology and Kretschmer's findings, the obesity bearing a strong resemblance to that described in his pyknic type.

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SUMMARY.

One hundred male cases of schizophrenia and of manic-depressive insanity were examined for morphological evidence of endocrine differences. It was found that the schizophrenics as a class showed relative hypogonadism, which had had its onset during the growth period, and that pituitary deficiency, of late onset, was frequent amongst the manic-depressives. These findings appeared to have some relationship to Kretschmer's morphological types.

I wish to express my thanks to Dr. A. Pool, Medical Superintendent of the South Yorkshire Mental Hospital, for his kind assistance and permission to publish these findings, and also all other members of the hospital staff who helped me in various capacities.

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IRRITABILITY AS A SYMPTOM IN MANIC-DEPRESSIVES.*

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IRRITABILITY is used in this paper in its popular sense as " the quality or state of being easily annoyed or excited to anger ", according to the definition of the *Oxford English Dictionary*. I leave it an open question how far other meanings of the word—especially " the condition of being excessively or morbidly excitable by or sensitive to the application of an external stimulus " —have anything to do with the definition of irritability as an affective state. A detailed description of the specific emotional quality of irritability would be of little help to someone who had never experienced it. Irritability might be characterized by its accelerated time factor (in contrast, e.g., to sadness, with its slow time factor), by its tension and proneness to release in outward expressions, and by its unrest. But all such characteristics are only descriptions of the same happening looked at from different angles.

It may seem important to recall the emotional reactions arising out of the irritable state : annoyance, anger, temper, rage, fury. Even if these reactions are suppressed, irritability remains and may even be stronger because the outburst is thus suppressed. It is the volitional inner struggle against these outbursts which gives irritability its special human touch. The reactions just mentioned have something in common in spite of their qualitative differences ; one can regard them as a sequence showing a quantitative increase of one common factor. This factor has been carefully studied in its biological implications by Cannon and his collaborators, so that we are especially fortunate in knowing at least the bodily correlates of the affect in question. Cannon, using anger and rage as synonyms for this common factor, emphasized the reflex character of the outburst. All the bodily adjustments he found in an enraged animal can, as he pointed out, be regarded as rendering the organism more efficient in struggle ; the more rapid heart-beat, the redistribution of the blood, the increase of red corpuscles in the circulation, the deeper ventilation of the lungs, the dilatation of the bronchioles, the liberation of sugar from the liver and the secretion of adrenaline. Discharges through the sympathetic

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division of the autonomic system cause these widespread visceral alterations accompanying rage. They can be called into operation in numerous conditions, of which asphyxial states and very vigorous muscular effort are of some interest here. Cannon was furthermore able to prove that a phylogenetically ancient part of the brain near the thalamic region is indispensable for getting the full rage response in cats. His and Marañon's observations gave no positive support to the theory of a peripheral source of emotional experience, as assumed by James and Lange in their assertion that the feeling of the visceral alterations constitutes the emotion itself.

Because of its familiarity in everyday experience, irritability is easily overlooked in morbid conditions. Although most text-books and monographs make cursory mention of irritability in manic-depressive psychoses, its frequency is seldom realized. Kahn found it in more than one-third of his 166 cases, and Lange agrees with him that this proportion is a minimum one. In Aubrey Lewis's cases of melancholia resentment was as frequent a reaction to depressive delusions as resignation. I was able to collect 25 irritable manic-depressives among the in-patients of the Maudsley Hospital within a short period.

However, if one pictures the typical depressive, with his sad mood retardation, feeling of insufficiency, hopelessness and anxiety, one feels rather disinclined to correlate all this psychologically with anger. And the same can be said of the manic with his hilarity, cheerfulness, flight of ideas, self-exaltation, vivacity and distractability. There is a contrast, just as there is between the choleric temperament on the one side and the sanguine or the melancholic on the other. But, as with most clinical descriptions, pure cases are rarities; the average clinical observations are blurred by all kinds of modifications. Since the typical affective syndrome has so relatively clear and simple a structure, the mere existence of modifications suggests a question as to the cause of these modifications.

I do not think it fruitful to relate this kind of irritability always to the existence of a "mixed state", as this term has been used in manic-depressive insanity. Most authors who have done so have followed Kraepelin. But Kraepelin accepted Specht's theory that irritability was to be regarded as a mixture of heightened self-consciousness and unpleasant moods; and if one remembers that Specht had derived "paranoia" too from similar emotional combinations, the rather arbitrary character of such speculations becomes evident. The time has, I think, passed when one could, as it were, mix the psychic colours on one's palette in order to explain morbid phenomena. Indeed the concept of mixed states itself has proved of practical use only in agitated depression and manic stupor.

In looking for another point of attack one might make use of the developmental aspect, and assume, according to the Freudian scheme, aggressive phases in infancy which can be related to adult irritability. I do not think this would disclose anything more than one knew beforehand. Irritability in

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depressions might, of course, easily be interpreted by stressing the aggressive-sadistic character of melancholia as Freud and Abraham have done. Even so, it is difficult to explain why irritability is so much more common in mania than in depression. Schilder, though a psycho-analyst, found the Freudian explanation unsatisfactory.

In studying the psychopathology of irritability one can start with the connection between irritability and fatigue and sleep. A certain degree of exhaustion or sleeplessness apparently leads to irritability in most normal persons. Barcroft's experiments have shown that insomnia as the effect of high altitudes makes people irritable irrespective of the amount of bodily exercise, even in a glass chamber with correspondingly diminished oxygen concentration. The remarkable fact that severe fatigue sometimes does not produce sleepiness, apathy or weakness (as sea-sickness regularly does), but leads to affective excitement and motor release, has most close points of contact with modern sleep theories like that of Hess. Hess, referring to Cannon's experiments, holds that rage is a counterpart to sleep as regards the interaction between the autonomic and the central nervous system. According to Hess parasympathetic activity of the autonomic system prevails in sleep. That is to say, the visceral alterations are in many respects just the opposite of those due to the sympathetic excitation in rage. Such a theory may enable us one day to find out under what conditions sleep is replaced by irritability—that is, the state in which reactions of anger or rage are easily provoked. Furthermore I think it justifiable to suppose that irritability after nervous exhaustion, i.e., in true neurasthenia, is perhaps due to insomnia and fatigue. Here is an obvious link with irritability in the sense of hypersensitivity, the latter being a typical neurasthenic symptom.

Normal irritability is connected at another point with the rhythm of sleeping and waking: it is experienced by many persons of every age each morning, apparently in many cases independently of the duration and depth of the preceding sleep. Gastro-intestinal influences are suggested by the soothing effect of breakfast or defæcation.

In the similar periodic swings of mood in the premenstrual phase or during the menstrual period of women physical causes of a different kind are at work. The continuous annoyance of tetanoid patients, especially after extirpation of the parathyroid glands, is a further instance.

Irritability is not the sole affective anomaly in all these states, but it plays an important part. Unfortunately no exact observations of periodic irritability in healthy people have, as far as I know, been made available. Even if the last word on this problem will have to be spoken by the biochemist, a collection of well-observed material, especially selected with regard to personality types, would be of the greatest interest. The fluctuations of mood just referred to are in all probability related to those more impressive daily fluctuations of mood found in manic-depressives.

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Irritability following intoxication, especially alcoholic, is, as far as we know, also more likely to occur in certain constitutional types. Some conditions in which it occurs are known ; but what we know is superficial, and only touches the problem from outside. Patients with severe head injuries or with non-traumatic epilepsy can become extremely irritated after a small dose of alcohol. Similar morbid emotional changes are observed without alcohol in epileptics as an aura, and as periodic and continuous states, the latter influencing the whole personality in respect to social conduct, etc. However, such patients in their periods of morbid irritation seek and find relief in consuming narcotics, preferably alcohol. Thus irritability can be soothed by the same toxic substance which excites it in other emotional conditions. This is not so strange as it seems at the first glance. As Wilder has expressed it in his " Ausgangswertgesetz " (law of the initial level), there are analogous variable effects of toxic substances on the sympathetic nervous system which can be excited or paralysed by the same substance, according to the level of excitement present at the moment of exhibiting the substance.

Iodoform and mercury are toxic substances which produce irritability if taken over a long period, apparently without individual differences. Kulkow in his description of mercurial poisoning in thermometer makers gives, however, a rather colourless picture of neurasthenia without considering sleep.

In recalling these well-known data about the psycho-somatic conditions of irritability, I have been trying to get some common ground from which we might turn more safely towards the psychological side of the phenomenon. Two typical social situations in which irritability arises may serve here as paradigms : irritability in cases of compensation neurosis and in persons of advanced age.

The first refers to a well-known figure in all countries where the system of insurance has been extensively developed as, e.g., in Germany. These neurotics, dominated by the idea of their right to compensation, have been the object of numerous studies. Such a patient lives a restless life of medical examinations, court sessions and investigations in hospitals. His mind is filled with the half-understood opinions and arguments of experts. Increasing discontent after repeated disappointments, lack of useful occupation and impotent ineffectiveness sum up the psychological situation. Permanent liability to outbursts of anger becomes the main feature even in persons who were entirely free from irritability before the key-event, i.e., the accident. Aubrey Lewis, in his investigation into neurotics with long-standing unemployment has found irritability a common complaint, probably due to psychological conditions similar to those just mentioned.

The analogous increase of irritability in advanced age can be understood in view of the loneliness, discontent, isolation and lack of adaptability of the elderly. That they should struggle for recognition in spite of their hopelessness is obvious. But it must of course be consid red here how far their customary

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insomnia contributes to the irritability on the one hand; on the other hand, irritability may be only the monotonous and rigid emotional reaction caused by senile or arteriosclerotic cerebral damage. Careful studies are lacking on this point, as so often the case in the psychophysiology of emotion. A constitutional factor, too, might be a possible explanation of senile irritability: personality features are often unveiled, as we know, in involutional mental changes.

All causal factors mentioned hitherto could, as one of my critics suggested, be summarized under the heading of "frustration". But such a summary designation would be a rather superficial manner of speech if compared with the numerous possible psycho-physiological connections revealed above.

Irritability as a leading quality in temperament has been dealt with by Kahn in his book on psychopathic personalities. It is the main feature, according to him, in two different types of temperament, which have, as I think, to be supplemented by a third. Kahn describes the last under the heading of "asthenic psychopaths" without giving irritability the attention it probably deserves. Kahn identifies his asthenics with people described in general medicine as "hypoplastics", cases of "infantilism", of constitutional nervousness. The concomitance between physical weakness, nervous sensitiveness and psychic exhaustibility is the main feature of these personalities. "They are easily tired and have a small capacity for accomplishment." They never have enough sleep or feel refreshed after it. The irritable depression which other people know as a brief morning phase lasts for a lifetime in these constitutional neurasthenics.

The second group of irritable persons, put by Kahn in the same series as the people of excitable and explosive temperament, can be called the sthenic counterpart of the first type. The irritability of the asthenic can be regarded as defence; the irritability of this type is more like attack. The general attitude in which it is embodied is more akin to that of the post-traumatic character change than to the behaviour of an exhausted climber in high altitudes. These explosive and irritable temperaments in question may perhaps all be post-traumatic in the sense that their anomaly is caused by brain injuries at birth or in early childhood. The proposal has been made in Germany to call them "epileptoids". There are obvious links with the quarrelsome and querulous type.

The third variety of constitutionally irritable people belongs to the "poikilothymic temperaments", which are, in Kahn's terms, the fundamental states of manic-depressive insanity. I had better quote a few sentences to show how Kahn understands irritability as a result "of the combination of components of the gloomy and euphoric make-up": "From the euphoric it obtains a bit of elation or excitement; from the gloomy a bit of blocking or inhibition. . . . However what . . . is characteristic, is the irritability of the poikilothymic engendered from opposite, and in a way conflicting

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temperamental elements. The stage of being torn to and fro between these elements conditions the duality in the irritable make-up. . . . They are . . . unable to find a way out." Such a theory, subtle as it sounds, is really only an elaboration of Kraepelin's mixing of affects. One could easily give a number of other reasons why euphoric or depressed temperaments should become irritable, none of them less plausible than Kahn's inner duality. Whatever the true explanation, undoubtedly there exist irritable temperaments of a kind closely related to the fundamental states of the manic-depressive.

Concluding this survey of the various conditions in which irritability appears, we have established it as a final outcome of different psychosomatic processes. Its phenomena are unambiguous and can easily be ascertained. It therefore deserves some interest, although it has no specific significance as a diagnostic sign in the still provisional nosological system of psychiatry.

Before applying the above to my observations, a few words must be said about the material. Lange was certainly right in criticizing Kahn's procedure, which consisted in collecting data about irritability by looking through a number of case-records. Kahn's work had nevertheless the practically important result of establishing the frequency of irritability as a premonitory symptom of both depression and mania. Diagnostic failures and misinterpretations due to neglect of this fact are indeed not uncommon. Difficulties and conflicts originating during this period of irritability are held responsible for causing the ensuing depression. Time and energy are uselessly spent in disposing of these difficulties, with the hope of a therapeutic effect which remains overdue.

In collecting my material, I sought the aid of the more experienced nurses of the hospital. The variety of material gathered with their help represents some typical cases and factors which will be dealt with under the aspects already outlined.

Considering first the problem of sleep and its influence on irritability, this material is neither large enough, nor could it be sufficiently carefully studied in this respect, to come to definite conclusions. One half of the twenty-five cases complained of disturbed sleep, and had received the usual sedative medicine, which did not seem to influence their irritability. One case of a long-standing depression was of special interest in this connection; he had been entirely sleepless during a depressive stupor in the course of his illness. But he became irritable afterwards when his insomnia improved. In order to estimate the sleep component exactly, one would have not only to measure the objective duration of sleep, but also its depth; for this the "aktogramme" method must be employed, which registers the movements in sleep, and has already been used successfully by Guttmann in manic-depressives.

It may be mentioned here that none of these patients was objectively retarded at the time when irritability was observed. Their subjective insufficiency and incapacity, so far as they complained of it, was always related to

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inner unrest, tension, feeling of "being hunted", etc. One half of the depressives spoke of this and showed impatience, discontent and restlessness in behaviour.

Only five patients reported daily fluctuations of mood, even if especially asked for them, and the objective reports did not throw much light on this problem. There was no general diminution of irritability during the course of the day, as far as objective observation goes. However, there were some striking examples:

One patient, æt. 25, who had previously had several short depressive phases told me that for as long as she could remember she felt like her old self only in the evening. She was always inclined to be irritable and got easily into a temper, especially during the first half of the day. But she never had any fluctuations of mood in relation to the menstrual cycle. Her irritability during the present depression was striking.

A middle-aged woman who suffered from a second attack of depression seemed a complementary case: she had been irritable during her periods since her youth. Irritability, which had been very marked from the beginning of her present depression, fluctuated in the following way: she felt rather free in the middle of the day, but worse in the morning and in the evening.

Such single cases, of course, can be only of heuristic value; they encourage one to collect similar material in larger quantity.

In the group of seven cases in which there was also an organic cerebral impairment the range of possible varieties was indicated. One of my patients was born by forceps after a difficult labour. Immediately after birth he had convulsions, which recurred on and off until he was 13. He was described as sensitive and self-conscious, but not irritable until, at the age of 27, his present depression began. After a suicidal attempt he came into hospital, where he showed marked daily fluctuations of mood (felt better in the evening) and a striking tendency to outbursts of anger.

In a patient aged 69 with high blood-pressure, but no clinical signs of dementia, the depression was coloured throughout by resentment, irritability and discontent. He was also mistrustful and slightly paranoid. But the latter trait was apparently constitutional; he lived as rather a hermit all his life, did not like company and trusted nobody.

A second patient of similar kind had no sign of hypertension. His irritability during depression was in marked contrast to his previous humorous and social personality. But he had had an accident ten years before, when he fractured his skull and remained unconscious for several days.

Irritability may have been of toxic origin in a man of 30, who had probably had cyclothymic fluctuations for years. He was mildly addicted to sedatives, and sometimes took a fair amount of alcohol. After a rather severe suicidal attempt he came into hospital, was very depressed, slept badly after the

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sedatives had been stopped, and was easily annoyed and inclined to outbursts of temper.

An endocrine complication, of possibly constitutional kind, may be responsible for the extreme irritability of a 19-year-old boy. He exhibited tetanoid hyperexcitability, marked by attacks of laboured breathing, with fainting and anxiety. These came on mostly in the evenings. He showed insomnia and slight retardation at the beginning of his depression, which slowly disappeared.

Speaking of those patients in whom irritability can be understood in the light of the psychological situation, I may mention that in six of my cases psycho-reactive factors played an important role in causing the depressive illness. The number of more reactive depressions—if such a distinction can be made, as against the more endogenous—is large among these irritable patients. That seems in accordance with the general clinical impression. Irritability was apparently due in all these cases to the difficult situation. Most of the patients were entangled in long-standing, rather insoluble difficulties concerned with sexual, family or professional life. Staying in hospital meant a delay, but not really a solution of the difficulties. The home situation was also filled with explosive elements; visits from their relatives led to outbursts of temper.

The situation in imminent old age, with loneliness, inability to work and helplessness accounted for the irritation in an elderly woman with involutional melancholia. She was continually angry with herself, impatient and discontented, entirely the opposite of her former personality. "I have lost my pluck; everybody is too good to me; I am a coward, worth nothing".

Turning to the question of constitutional peculiarities which might have contributed to irritability in my manic-depressives, I found seven patients of a rather similar type of personality; one might call them sensitive asthenics. They were shy, very dependent, ambitious, but exhaustible, unsocial and slightly mistrustful. Irritability was not a prominent feature in their original character, but became very marked during their depressive illness.

An entirely different type of temperament was represented by the case of a 19-year-old girl who was the daughter of a drunkard and a neurasthenic mother. She had tempers as a child, and had been always depressed and irritable during the first two days of her menstrual periods. She was otherwise regarded as cheerful and fond of company, but easily roused. In her first depression, two years ago, she showed some claustrophobia. This time she had numerous clashes in the hospital because of her excitability and anger on trivial occasions: "If one of the patients talks too much at tea-time, I run out of the room and bang the door."

There were several cases of similar kind in which irritability was a constant feature of personality, and could often be found in other members of the patient's family. This feature became more marked in the patient during

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the depressive or manic attack. But habitual irritability can also disappear during a depression, as I observed in one patient who was, however, slightly retarded and entirely absorbed in self-reproach.

When approaching constitutions more or less akin to the fundamental states of the manic-depressive illness, the explanation of irritability becomes more difficult. I saw, e.g., an engine-driver, aged 42, who had undoubtedly been of depressive constitution all his life, a solemn, lonely, very conscientious man. In his depression, which has now lasted two years, he has become more and more irritable ; he feels angry against himself, because he is unable to control himself, but his annoyance is also directed against other people. I do not know how to account for this irritability except by relating it to his very labile blood-pressure, which sometimes rose up to 160 under emotional influences. This again might be a constitutional factor.

One of my patients was an extraverted syntonic character, corresponding in every respect to Kahn's poikilothymic temperament. In his forty-ninth year he became depressed for the fourth time in his life, being rather hypomanic in the time between attacks. He then got on well with everybody, had numerous hobbies and belonged to many clubs. The depressive attack, which has now lasted six months, began with a state of severe retardation. When this improved he developed the present state. He is impatient, distrustful, easily irritated and restless.

Finally, three patients exhibiting irritability during manic attacks may be mentioned. All had, of course, severe insomnia. One of the cases who was manic for the second time at the age of 35 became slightly confused at the peak of his manic excitement, and had outbursts of anger with aggressive behaviour. The two others had familial and habitual tendencies to irritation, like those found in the cases of depression. No typical mixed state was contained in my material.

In conclusion, the result of my inquiries into the frequency of the above factors is given in the following table. The corresponding figures, as obtained from twenty-five manic-depressives who did not show irritability during their psychoses, are given for comparison. Only the importance of constitutional factors (7 and 8) is confirmed by this numerical method, the value of which should not be over-estimated in view of the low number of patients and other fallacies. A symptom like irritability, which is a final outcome of several diverse processes, may only become apparent when more than one of these factors, or of other as yet unknown conditions, occur in the same patient. It would perhaps be more illuminating if the two columns could be compared with figures obtained from a series of irritable people without manic-depressive psychosis.

The much higher frequency of hypersensitivity to external stimuli (especially noises) in non-irritable patients (9) is difficult to account for, and calls for further investigation.

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Findings.

	Among 25 irritable manic-depressives.	Among 25 average manic-depressives.
(1) Habitual anomalies of sleep	9	8
(2) Daily fluctuations of mood in psychoses	5	10
(3) Premenstrual or menstrual fluctuations of mood	4	5
(4) Brain injury, encephalitis, etc.	4	5
(5) Alcoholism and other drug addiction	2	1
(6) Anomalies of the circulatory system	3	4
(7) Constitutional irritability, irritability of family	9	5
(8) Constitutional sensitive asthenics	7	4
(9) Hypersensitivity (habitual and during psychosis)	2	12

SUMMARY.

Irritability (in the sense of "the quality or state of being easily annoyed or excited to anger") is considered in its psychobiological aspects in the first part of this paper. In the second part these considerations are applied to twenty-five patients who exhibited irritability during a mental disorder of the manic-depressive type.

The author wishes to express his gratitude to Prof. Mapother for his kind permission to make use of the clinical material of the Maudsley Hospital.

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ATTEMPTED SUICIDE: AN INVESTIGATION.

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PROVISION for the observation of cases of mental disturbance occurring in the administrative and the user area of the Liverpool Corporation is made in one of the city institutions, Smithdown Road Hospital. In this large general hospital are located two special divisions, containing 180 beds, one for female and one for male patients, and to the wards of these divisions are receivable all cases of attempted suicide occurring in Liverpool.

It is probable that the great majority of such cases occurring in the area reach this hospital. Severe and urgent cases may be admitted to the nearest hospital, but a large proportion of these, if they survive, are transferred when able to be moved. No doubt a number of cases, especially those of mild degree or of persons of ample means, never reach hospital. These special divisions, however, do receive all types and degrees, and the total can be taken as representative of this problem in Liverpool. The area is an urban one, a great seaport, and the population includes many alien nationalities.

The great majority of the patients are committed to hospital on Orders for Temporary Detention (Section 20), completed by police or receiving officers, a small proportion on the certificates of outside doctors, in other hospitals or in practice. In these latter cases action as to detention is taken by the medical superintendent under Section 24 of the Lunacy Act, where he considers such action is necessary.

The concentration of all such cases from a limited but heavily populated area provides an excellent opportunity for the study of attempted suicide. The material and social conditions are known or easily investigated, and relatives, friends, relieving officers, police and probation officers are usually available to provide information.

It was therefore decided to make an investigation into the condition, history and circumstances of the patients who had been admitted to this hospital on account of having attempted suicide, the object being to discover the factors which had led to such action. Such an inquiry obviously entails a great deal of work in the detailed investigation of each patient, the

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interviewing of relatives, friends and other informants. It was decided to limit the number to 100 cases, taking 50 consecutive admissions of each sex. Information would be required as to each patient's social and economic state, the personal history (including health and occupational history), family history, the circumstances leading up to the attempt, the ascertainment of stresses, the mental and physical state, the method and severity of the attempt, the history of any previous attempts, the attitude of the patient on initial recovery, the treatment, result and disposal of the patient. No effort is made to consider the legal aspects of the problem, nor its psychological mechanisms; nor could any reliable estimate of the incidence of attempted suicide be made.

In such a small number of cases certain factual details would have little significance, and it was known that the hospital records contained some information regarding all such admissions over a number of years. It was possible to obtain in each case the age of the patient, the date of the attempt, the method employed, and the disposal of the patient. It was therefore decided to obtain these data in respect of all cases of attempted suicide admitted to the hospital during the four years 1932 to 1935.

The interest and value of this information would be enhanced by comparison with similar data regarding actual suicide in the Liverpool area. Dr. G. Cecil Mort, Barrister-at-Law, H.M. Coroner for the City of Liverpool, kindly consented to supply this information concerning the suicides dealt with at the Liverpool City Coroner's Court during the four years 1932 to 1935. I am indebted to him and his clerical assistants for this time-consuming task, so generously and voluntarily undertaken.

As will be seen in Table I, the total number of cases of attempted suicide admitted to the hospital during the four years was 656, and the total number of suicides dealt with at the Liverpool City Coroner's Court during the same period was 399.

The numbers of attempted suicides given here cannot be taken as the total incidence for Liverpool, nor do they show the numerical relation of

Table I.

Year.	Number of cases of attempted suicide admitted to Smithdown Road Hospital.		Number of suicides dealt with at the Liverpool City Coroner's Court.	
	Males.	Females.	Males.	Females.
1932	99	75	81	25
1933	85	68	78	34
1934	96	73	66	32
1935	78	82	64	19
Totals	358	298	289	110

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attempted to actual suicide, because the area served by the hospital differs, to some extent, from that of the City Coroner's Court. But the social conditions are closely approximate. It will be noted that in attempted suicide the ratio of female to male cases is 5 to 6, whereas in suicide it is less than 2 to 5. (*N.B.*—This ratio for attempted suicide is probably as near as can be ascertained in comparing the two sexes.) The Registrar-General's Review for England and Wales (1) shows for suicide a fairly constant similar relation of the sexes for many years, there being more than twice as many males as females, although the totals had risen from 3,740 in 1924 to 4,550 in 1934.

Age-Incidence.

Table II.

	Age.												
	< 15.	15–19.	20–24.	25–29.	30–34.	35–39.	40–44.	45–49.	50–54.	55–59.	60–64.	65–69.	70+.
Attempted suicide:													
Females . .	2	27	52	35	31	25	31	24	22	18	13	9	9
Males . .	0	11	32	47	43	31	34	41	35	24	27	16	17
Suicide:													
Females . .	1	3	5	8	9	11	10	15	11	17	11	5	4
Males . .	1	7	19	13	15	19	25	31	35	42	33	25	24

In women the incidence of attempted suicide is at its highest in the quinquennium 20–24, namely 52 cases, followed by 35 cases for the ages 25–29. After 45 there is a steady decline, with no special increase for the climacteric period.

In men there is a more even distribution at the various age-periods, the maximum occurring during the decennium 25–34, as was also observed by East (2) in 1,000 male cases. The decline in incidence begins after 49.

The figures for each sex show a close similarity to those found by Lendrum (4) in analysing 1,000 cases of attempted suicide between 1927 and 1930.

In the Liverpool figures for suicide the number of female cases is small, but shows a steady rise to middle life, and with the males we find a similar distribution, both sexes showing a maximum at the 55–59 period.

In the figures for suicide in England and Wales in 1933 and 1934 (1) the maximum incidence in males in both years is at the 55–64 decennium, and in females at 45–54. And whereas from the ages of 20–44 the proportion of males to females is less than 2 to 1, at 55–64 in each year it is more than 3 to 1.

There is no doubt that, as was observed by Morselli (3), the tendency to

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suicide occurs at an earlier age in women than in men. This observation applies also to attempted suicide, which furthermore appears to be more prevalent at earlier ages than is the case with suicide.

The frequency of attempted suicide in females under the age of thirty is probably due to the frequency of amatory disturbance in unmarried girls and the disappointments and quarrels of early married life. When we compare these figures with the small incidence of actual suicide during the same period, doubt is cast on the authenticity of these attempts.

Monthly Incidence.

Table III.

	Month.											
	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Attempted suicide:												
Females . .	20	14	23	27	16	42	34	24	29	20	24	25
Males . .	24	26	19	34	26	34	36	43	35	23	25	33
Suicide:												
Females . .	8	11	9	4	11	11	6	10	4	10	12	13
Males . .	14	26	23	29	29	27	31	23	23	23	19	22

The maximum monthly incidence of attempted suicide in males was in August (43) and in females in June (42). For the six months April to September in the four years there were 172 female and 208 male cases, whereas for the six months October to March there were 126 female and 150 male cases.

It is a generally accepted fact, shown by official statistics, that, in the Northern Hemisphere, suicide is more prevalent during the warmer months of the year. This would appear to be true also for attempted suicide.

In the figures for suicide given here, males show a preponderance during the spring and summer months, but the reverse is seen in the female cases. Here, however, the numbers are small.

Various theories have been propounded to explain the seasonal incidence of suicide, but none that has received general acceptance. The suggestion is here offered, that whereas in the colder months of the year the members of a community live in close contact, in the longer and warmer days they tend to scatter, and the patient may feel more acutely his difficulties and his isolation.

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Methods Employed.

Table IV.

Method.	Attempted suicide.				Suicide.			
	Males.		Females.		Males.		Females.	
	No.	%.	No.	%.	No.	%.	No.	%.
Coal-gas poisoning	123	34·3	110	36·9	135	46·7	76	69·1
Cut throat . .	75	20·9	20	6·7	30	10·4	2	1·8
Liquid and corrosive poisons	68	18·9	106	35·6	34	11·7	20	18·2
Sedative poisons .	6	1·7	13	4·4	4	1·4	1	·9
Immersion . .	26	7·3	25	8·4	14	4·8	2	1·8
Cut wrists, stabbing, etc.	10	2·8	4	1·3	1	·3	none	..
Strangulation, hanging	19	5·3	13	4·4	45	15·8	5	4·5
Jumping from heights or in front of vehicles	19	5·3	7	2·3	22	7·6	4	3·6
Gunshot . .	2	·6	none	..	4	1·4	none	..
Miscellaneous .	10	2·8	..	..	..	..	..	..

In the Coroner's statistics a number of cases of death from immersion were not included because there had been no evidence to show how they came to be in the water. No doubt a proportion of these were cases of suicide.

In suicide the sedative poisons used were morphine, nicotine and veronal; in attempted suicide, aspirin (11), dial (3), luminal (2), veronal, atropine and chlorodyne.

Taking both sexes together it will be seen that in these four years the commonest method of attempted suicide is by coal-gas poisoning

It will be noted, however, that while attempts in both sexes by coal gas are approximately equal, in cut throats men exceed women by nearly 3 to 1. In women, attempts by both corrosive and sedative poisons are twice as common as with men. Immersion is approximately equal in both sexes, but in the other methods men exceed women to some extent, although it is noted that here the numbers are small.

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The records show a fairly constant yearly incidence for each method. For instance, the number of gas-poisoning cases in men for each of the four years was 24, 30, 26, 30, and in women, 33, 36, 25, 29. For cut throats in men, 23, 15, 20, 17, and in women, 7, 5, 4, 4. And so on for the other methods.

Some relation of the method used to the age of the individual was also noted. Gassing occurs mostly in persons below the age of 35, attempts by liquid and corrosive poisons a little later, and cut throats in the latter half of life. The last method accounted for most of the attempts made by elderly men.

Attempts by immersion occurred principally during the warmer months of the year, but otherwise the method of election had no special seasonal incidence.

The varieties of corrosive poisons used were numerous, and a precise statement of their nature was often difficult to obtain. In male cases liniments of various kinds were the most frequent (12 cases), followed by lysol (7), oxalic acid (6), iodine (6), salts of lemon (4), sulphuric acid, carbolic acid, rat poison, ammonia (each 3), and various disinfectants, lotions, acids, etc.

The attempts were mostly by some single method, occasionally more than one was used. As a rule one of these attempts predominated, the other being tentative or slight, and here only the major attempt is cited.

It is interesting to compare here the prevalence of the various methods in these years with those found by Norwood East (5) among male cases in Brixton Prison during the years 1907 to 1910. In his series of 1,000 cases there were only 2% by coal-gas poisoning, but 32% by other poisons (including 9·3% by oxalic acid) and 19·3% by drowning. Cut throats in that series totalled approximately 27%. These facts show the change that has taken place in the choice of means of self-destruction, although it must be noted that the cases were a selected group in that they included only male suicidals concerning whom police action had been taken.

The reason for the disparity in the methods of the two sexes is not far to seek. It may reasonably be expected that an individual will use the means with which he or she is most familiar and which is near at hand. The razor is predominantly a male instrument; the woman in the home has charge of disinfectants, cleaning materials, and medicines as a rule. It may generally be said that the male will more readily use violent methods. There are other factors concerned with the mental state which have a bearing on this point and will be discussed later.

When we consider the methods that have been used in actual suicide, we find in the Liverpool figures considerable disparity in comparison with those used in attempted suicide. Here, however, coal-gas poisoning is by far the most common method, in females accounting for nearly 70% of all suicides. In other types of poisoning, and in cut throat, the incidence for suicide is only about half that of attempted suicide. Hanging and strangulation was more frequent in suicide than in attempted suicide.

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The figures for the country as a whole (1) show that coal-gas poisoning is the commonest method with both sexes, and a remarkable rise in its incidence during the past few years. In 1924 there were 585 deaths from this cause, and in 1925, 762, while in 1933 there were 1,732, and in 1934, 1,864. Similarly, suicides from other poisons rose from 413 and 466 in 1924 and 1925 to 816 and 749 in 1933 and 1934. There has been some increase in suicide by jumping from heights and in front of vehicles, but those by drowning, hanging, cutting and piercing instruments and gunshot show no material change.

Suicidal attempts by cut throat and by corrosive poisons account for about 40% of the total in each sex. In suicide these methods are responsible for only 20%. The reason for this difference would appear to be mainly due to the fact that both these methods are uncertain and liable to miscalculation. Coal-gas poisoning, on the other hand, if the attempt is determined and no interference occurs, can easily be made certain.

Disposal of Patients.

Table V.

	Males.	Females.	Total.
To friends	183	193	376
To police	66	37	103
To mental hospitals	43 (12%)	27 (9%)	70 (10·7% approx.)
To other wards, hospitals or institutions	40	28	68
Died	26	13	39

Some clarification of the terms used here is necessary. When a patient is discharged from hospital it is necessary that he should have a home to go to, and relatives or friends who will accept some responsibility for his care. Discharge to friends does not necessarily mean that the patient has gained complete normality, but that the mental and/or physical state is such that retention in hospital is no longer considered essential. A certain proportion of cases are taken out of hospital against medical advice (provided no police action is contemplated), the friends assuming all responsibility.

Not infrequently it happens that the patient has no friends or relatives, or that these refuse to accept him. Such cases as a rule are transferred to an appropriate institution. Other cases, requiring further treatment for medical or surgical conditions, are transferred to general wards or other hospitals.

A large percentage of cases are committed to hospital by the police on the completion of temporary detention orders. In a proportion of these cases they express the intention of taking action when the patient is fit to

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leave hospital, and the patient is discharged to them. When a charge is brought, however, it is extremely rare that any sentence is imposed.

No attempt is made to classify the causes of death in the patients under observation. Many are suffering from chronic or advanced forms of physical illness, and it is often very difficult to assess to what extent, if any, the suicidal attempt has contributed to the patient's end.

It will be seen that nearly 11% of the total admissions are certified and transferred to mental hospitals. This is a significant figure, especially when it is realized that many who have been mentally disordered on admission quickly clear up, and others are discharged to friends against advice.

Analysis of the methods employed by patients later sent to mental hospitals shows certain features of interest.

Table VI.

Method.	Total.	To mental hospitals.	Percentage.
Coal-gas poisoning	233	10	4+
Liquid, corrosive and sedative poisons	193	8	4
Cut throat	95	23	24
Immersion	51	11	22
Hanging and strangulation	32	9	28
Jumping from heights, in front of vehicles	26	2	8
Cut wrists, stabs, etc.	14	6	43

The small number of poisoning cases that it was found necessary to send to mental hospital compares in striking fashion with the large percentage of what might be called the more violent methods. The numbers here are comparatively small, but the incidence of certified cases is consistently high with the exception of those who have attempted self-destruction by leaping from heights or by throwing themselves in front of vehicles. It may be that in this group there are many whose attempt has been more of the nature of a demonstration than a serious attempt at suicide.

The conclusion has been reached, however, that when methods used are such as would, in the event of failure, leave disfigurement or disablement, then the mental state must be viewed with suspicion.

Detailed Investigation of One Hundred Consecutive Cases of Attempted Suicide.

These cases were admitted to hospital during the period September to April, and the number of male and female patients reached totals of 50

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within about one week of each other, that is to say that the incidence of patients of both sexes was about equal, whereas as a rule males preponderate in the proportion of about 6 to 5. The rate of admission was approximately normal for the hospital; there was no unusual prevalence or otherwise of attempted suicide during the period under review.

On account of the small numbers, much of the information of a statistical nature given here can have little general value, but it is included for completeness, and in order to show in as much detail as possible the material under consideration.

Method of Admission.

The majority of cases were admitted to the hospital under Section 20 of the Lunacy Act, the Temporary Detention Order being completed by the appropriate relieving officer or a police-constable.

Of the female patients, 43 were admitted under Section 20 (27 by police and 16 by relieving officers) and 7 on doctors' certificates. Forty-one males were admitted under Section 20 (31 by police and 10 by relieving officers), and 9 on doctors' certificates.

Age-Incidence.

In the female group there were 19 cases between the ages of 15 and 24, 9 under 20 and 10 over. Of the males, however, there were only 5 cases in this age-group, the greatest incidence being in the quinquennium 30–34, viz., 10, followed by 8 between the ages 45–49. There were no female cases above the age of 70 and only one male.

These figures show some comparable relationship to the figures for the larger total previously considered. A notable point in this group is the large percentage of suicidal attempts in unmarried girls below the age of 25.

Marital Status.

	Married, with children.	Married, no children.	Widows, widowers.	Single.
Females	20	3	2	25
Males	19	6	6	19

Religion.

Sixty-three patients were said to be Protestant and 37 Roman Catholic. No comment is made on these figures, except to state that frequently religion appeared to be little more than a name, and creed merely a necessary label.

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Occupations.

Many of the patients had been unemployed prior to admission to hospital, and some had gone from one type of work to another. The information given here is of the usual or latest type of work. Twenty-five of the women patients had been engaged in household duties in their own homes; 8 were domestic servants; 4 had been living at home with no employment, maintained by their parents or guardians, and 1 had been an inmate of an institution for several years. There were 3 factory hands, 2 machinists, 2 shop assistants, and the remaining occupations were nurse, theatre attendant, shop-keeper, bag mender and waitress.

Of the males, 23 were labourers (including dock labourers) and 6 were seafarers. The remainder included clerks (2), machinists (2), dairymen (2), newsvendors (2), and one each of the following: merchant, dentist, school teacher, ship's scaler, tram-driver, coalman, foundryman, cabinet-maker, cinema manager, wood-turner, coachman, checker, glazier.

Employment and Means of Subsistence.

Of the females, 22 were housewives supported by their husbands, 10 were living with or were supported by parents, 10 were in employment and thus maintaining themselves; 7 were unemployed and subsisted on pension, unemployment or public assistance, and 1 was drawing National Health Insurance benefit.

Fifteen males were employed and living on their earnings, and 3 were living on capital or were supported by parents; 26 were unemployed and subsisting on unemployment or public assistance, pensions, etc., and 6 were drawing National Health Insurance benefit.

Economic Position.

Financial insecurity or difficulty is commonly considered of primary importance in determining the mental attitude of those who commit suicide or attempt to do so. The assessment of any individual's economic position can only be made by a consideration of numerous factors, such as income, size of family, essential expenditure, social status, standard of living, habits, and so on. In this series, every case was investigated as to income and its apportionment, the history and environment of the patient, his or her ability in the management of domestic affairs, the existence of debts and the urgency of repayment.

The main object in this connection was to discover, not whether the patient was living in poverty or comfort, but whether or not the financial position was

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one of stress to the individual. In analysing this question it was decided that the cases could be placed in three groups, viz., those who enjoyed adequate financial means (i.e., adequate to their needs and custom), those who lived in poverty, but without distress, and those who were under definite economic stress. In this last group are cases which were above the poverty line, but who by reason of abnormal expenditure, bad management, borrowing, gambling (especially in wives unknown to their husbands), were suffering definite anxiety on financial grounds.

Of the females, 16 had adequate means, 26 were living in poverty but without distress, and 8 were labouring under economic stress.

Of the males, the respective totals were 12, 28 and 10.

It can be said that 18% of this series of cases were subjected to economic stress.

Personal and Occupational History.

In each case as much information as possible was obtained as to the patient's early life, environment, education, occupation and personal experiences. In some cases a great deal was elicited, often of a reliable nature; in others the information was either scanty or unreliable, or both. In these circumstances, particulars of this type could only be of value in a general way, i.e., in helping one to form an idea of individual personalities and environmental circumstances.

Health History.

Here the information was, as a rule, more readily obtained, and it was often possible to verify the statements made. In the female group, 28 gave a good health history, having suffered from no illnesses other than minor complaints, and having had no prolonged period of invalidism. One had previously been an inmate of a mental hospital; 8 had persistently suffered from "nerves", i.e., psychological disorder of some kind and degree; 6 had had persistent physical ill-health, 2 were post-encephalitics, 1 an epileptic, another a chlorodyne addict, and 3 gave a history of psychological disorder in the past.

In the male group 18 gave a featureless history; 3 had previously been in mental hospitals; 7 had been suffering from psychological disorder, while 8 gave a history of similar trouble in the past; 1 was an epileptic, and 13 complained of persistent physical ill-health. These last included a variety of conditions, such as carcinoma of the liver, progressive loss of sight, valvular disease of the heart, duodenal and gastric ulcer, tabes dorsalis, dementia paralytica, chronic bronchitis, chronic eczema, pulmonary tuberculosis, gastritis.

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Family History.

Little reliance could be placed on the information received in this connection either from patients, relatives or friends. Two of the women were of illegitimate birth, and 6 were known to have an unfavourable family history as compared with 7 of the male cases.

Methods of Attempt.

The frequency of the methods used shows a close approximation to Table IV.

	Females.	Males.		Females.	Males.
Coal-gas	13	17	Cut wrists, stabs	2	3
Corrosive poisons	18	11	Immersion	5	2
Sedative poisons	3	*nil*	Hanging, strangulation	3	3
Cut throat	5	14	Jumping from heights	1	*nil*

Previous Attempts.

Two women and 10 men in this series of cases had made previous attempts at suicide.

Severity of Attempts.

This is difficult to assess in view of the fact that often the most determined suicidal attempt is discovered at the critical moment, so that the attempt is either frustrated or limited. The important question from the psychological-prognostic point of view is whether the attempt is a determined one. This is often a difficult matter to elucidate, especially on account of the varying mental states and on account of the mental attitude of the patient who finds that his effort has failed. Nevertheless it is a question about which the doctor has to come to a decision.

In this series it was decided that in 16 females and 14 males the attempt had been slight, with no underlying determination to terminate life. The remainder were determined and real attempts at suicide. The results in the female cases were severe in 15 cases, moderate in 10 and slight in 9. Of the males there were 14 severe cases, 7 moderate and 15 slight.

Physical State on Admission.

Here one is not concerned with the effects, mostly temporary, of the efforts at self-injury, but with the general state of bodily health at the time of the attempt of those who have attempted suicide. The findings given here are the results of routine physical examination or of known disorders. It should be mentioned that a Wassermann test of the blood is made in all patients admitted to the special divisions of the hospital.

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Thirty-one of the female patients appeared to be in normal physical health, and 8 others, although showing no signs of organic disorder, were of notably poor nutrition. One patient was pregnant though unmarried, and another, a young married woman, had had a Cæsarean section with a live healthy baby one week prior to her attempt. One had syphilis with a positive Wassermann reaction, 2 were post-encephalitics, 1 was a long-standing case of epilepsy, and another had severe and regular attacks of migraine. The remaining 4 patients suffered from carcinoma of the peritoneum, chronic nephritis (both died in hospital), mitral stenosis, severe anæmia of secondary type.

Of the males, 39 were in good general health on admission and there was 1 epileptic. Of the remaining 10, 3 were suffering from neuro-syphilis (general paralysis of the insane, tabo-paresis, tabes dorsalis), and the rest were in poor health, 3 being extremely debilitated. Diagnoses were made of carcinoma of the liver, gastric and duodenal ulcers, mitral stenosis and incompetence, influenza.

Mental States on Admission.

Lacking absolute standards, the assessment of mental states must be a matter depending on the judgment of the individual examiner. The information here given was the result of examination on admission to the hospital, or at the earliest time after the masking effects, if any, of the suicidal attempt had passed away. In a number of cases the abnormal mental symptoms quickly cleared up, but it can be assumed generally, that the mental state on admission was the mental state at the time of the attempt.

Of the female patients, 12 showed no evidence of any mental abnormality elicitable by examination. They showed no clouding of consciousness, deterioration of intellectual functions, emotional disturbance or abnormal ideas. Twelve were psychotic ; 11 were depressed, but not to extreme degree and still retained insight. The remaining 15 were neither insane nor mentally normal, and can be described as being chronically unstable persons of poor stamina, 1 being a post-encephalitic and 1 an epileptic.

Two of the males showed no departure from normality ; 20 were psychotic. Five showed only depression, 1 was under the influence of alcohol, and 22 were of the persistently unstable type with poor resistance to stress.

Classification of the types of mental disorder, often in the early stages and sometimes of a transient nature, where the period of observation is comparatively short, is a difficult task. The groupings shown below give, however, a general idea of the various types.

Seven of the female psychotics were melancholic, 1 an acute mania, 2 schizophrenic, 1 a senile dement and 1 a paraphrenia.

Five males were melancholic and 1 acutely maniacal. There were 3

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dementias of senile type, 1 of dementia paralytica and 1 of tabo-paresis. Three were schizophrenics, 1 a delirium tremens and 2 were confusional states, one of these being associated with epilepsy. Three other patients had persecutory delusions and hallucinations of uncertain clinical type.

Results and Disposal.

Four of the female patients died, and 4 were still mentally disordered at the end of their period in hospital. Twenty-two appeared to be mentally normal, and 20 showed no gross mental abnormalities, but were considered to be unstable. Of the 46 who survived, 4 were certified and transferred to the county mental hospital for more permanent detention. Seven were discharged to the police, 32 to their relatives or friends, 3 to other wards and institutions.

Three of the male patients died and 15 were still mentally disordered. Nine appeared to be mentally normal and 23 were quiescent but unstable. On discharge, 9 were certified and transferred to mental hospital, 4 were transferred to other wards or institutions, 17 were discharged to the police and 17 to relatives or friends.

The 7 cases transferred to other wards or institutions were patients who required convalescent treatment or treatment of the physical state when the mental state warranted discharge from supervision, or when no relatives or friends were available.

In a certain number of cases patients were discharged to friends against advice when they would still have benefited by hospital treatment, but their condition was not sufficiently serious to insist on retention.

In some cases the police intimated that they wished to take charge of the patient when the latter had recovered sufficiently to answer a charge.

Causes of the Attempts.

Every case is an individual problem with characteristics peculiar to that individual. In each case there is a train of events leading up to the attempt. In some cases investigation shows this plainly, in others it is obscure. There is, however, always a final circumstance which determines the event. These determining causes are the causes commonly given in explaining suicide, such as mental or physical disorder or domestic and economic troubles, unemployment and the like. The common practice of ascribing the cause of suicide to some particular stress is no doubt satisfying to those concerned in giving a feeling of completeness and conclusion to the matter. But such explanations as unemployment, business worries or domestic difficulties can surely be no satisfactory reason why isolated members of the community should endeavour to terminate their lives. On the other hand, a real and

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complete understanding of the causes for such action would necessitate so prolonged and detailed a study of the individual as is impossible in practice. This might be carried out in a very small number of cases, but would not necessarily have a general bearing on the problem, unless it could be definitely established that there is a common factor in the mental development of such individuals.

In the present series of cases the effort was made to discover not only the reasons why the patient decided to attempt self-destruction, but also the circumstances which had led up to such a reaction. From the history given by patients, from observations made by relatives and friends and from other sources, it was noted that a very large proportion of cases had shown characteristics of personality varying in definite degree from the normal. It became increasingly evident that these factors, although playing no part in the individual's conscious determination of his behaviour, yet were of the greatest importance in influencing the quality of his reactions. Many patients had made suicidal attempts for reasons which to the average individual would be inadequate, and which could be understood only when considered in relation to the personality and history of the patient. In other words, it was realized that in addition to the determining causes there were what I propose to call *conditioning* causes. These causes include characteristics of personality showing definite deviation from the normal (or average), and physical states which were the primary cause of changes in the mental attitude. No effort has been made to investigate their origins, but merely to record the personal characteristics which have coloured the individual lives.

It might be contended, and with reason, that in investigating a consecutive series of cases admitted to hospital on account of attempted suicide, one may be dealing not solely with cases who have attempted self-destruction, but also with a proportion whose motive was essentially different, viz., to produce a similar effect in order to gain personal ends. That is to say, there may be cases whose actions are essentially hysterical, or comparable to the self-infliction of disabling wounds. A decision on this point, especially after the event, is always a difficult one, and it must be remembered that the great majority of patients are notified to the police, relieving officers or doctors by close relatives, whose natural tendency, were it not for fear and anxiety for the patient, would be to conceal the occurrence.

In Table VII is given a list of the causes which have precipitated the attempts. In some cases more than one have been operative in the same individual, as, for instance, domestic stress, alcohol, ill-health, unemployment and economic stress.

It will be seen that the commonest cause is mental disorder. It is not suggested that other factors have played no part, for indeed the psychosis must have been the result of faulty reaction to internal or external stresses. But here the immediate cause of the action was the abnormal state of the

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PRECIPITATING CAUSES OF SUICIDAL ATTEMPTS.

Table VII.

Cause.	Females.	Males.
Mental disorder	12	20
Domestic stress	18	12
Business or economic stress	8	12
Physical ill-health	8	5
Alcohol	5	8
Unemployment	none	6
Amatory disturbances	9	none
Old age and loneliness	1	1
Impending police charge, or fear of	1	2
Pregnancy, or fear of	2	..
Bravado	..	1

patient's mind, whatever the cause of that abnormality may have been. It was notable that in almost every case some previous abnormality had been noted. In the males, 8 of these patients had been of abnormally shut-in personality, 3 had histories of erratic and unstable behaviour, 3 had suffered from serious physical ill-health, and there was 1 case of general paralysis of the insane and 1 of tabo-paresis. One was in a condition of delirium tremens and 1 of post-epileptic mania. Of the female patients, 7 had previously shown abnormalities of personality or temperament.

The term " domestic stress " is somewhat vague, but is meant to include such circumstances as deaths in the family, quarrels and disharmony on various accounts, such as religion, inconstancy, maintenance, etc. It is not surprising that the numbers under this heading should be comparatively large when the emotional relationships of family life have so many aspects. As might be expected, the effects were more frequent in women, because to women life as a rule is focused domestically. Of the 18 female cases the majority had previously shown signs of unusual personality or unstable temperament, but in 7 cases the individuals appeared to have been average personalities and their action the result only of domestic stress. Four of these patients had made severe and very determined attempts following cruelty, neglect or unfaithfulness on the part of their husbands. In the male patients domestic stress was the cause of attempts only in association with other stresses, or in individuals with histories of inferior psychological stamina or make-up.

Business or economic stress appeared but rarely to be a primary cause of attempts, but influenced the action of 8 male cases of abnormally shut-in type, and in 3 of these may have precipitated mental disorder.

Where physical ill-health is given as a cause of the attempt it is meant that the patient's attitude to his physical condition has influenced his or her action.

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Two cases only were influenced by pain. One man suffering from general paralysis of the insane was becoming blind and this disability was the determining cause of his action, associated with the fact that he was mentally disordered. A girl, suffering from epilepsy, made her attempt because she realized her prognosis and felt herself a burden on her family.

Alcohol appeared to be only a contributory factor in 11 of the 13 cases. One of the remaining 2 was a case of delirium tremens.

Unemployment similarly was in no instance a primary cause, but had influenced 3 men who were of shut-in type, 2 who were feeble-minded and 1 an epileptic.

Love affairs which were not progressing satisfactorily were responsible for the attempts in 8 single females ranging between the ages of 17 and 23 and one aged 36. The last was a person of poor mental and moral type, and her action, which was slight, was carried out under the influence of alcohol. Three of the others had a history of temperamental instability, but in the remaining 5 no other factor could be elicited. One was four months pregnant and had quarrelled with her lover, and another feared pregnancy. The attempts in these two cases were both severe and determined; in the others they were slight.

A man of 74 had lived alone for twenty years in one room, with no friends and no outside interests. A woman of 68 had lived a similar life for twelve years. With decreasing vitality and nothing to live for, self-destruction seemed to them a logical solution of their problems.

Fear of impending charge by the police was a precipitating cause of attempts in 3 cases. One was a girl of 17 who lived with her family in constant disharmony. She was in daily domestic service, had stolen from her employer, and her attempt, which was slight (by drinking iodine), was probably made as a temporary solution of a difficult position. A man of 47, who had for years been of solitary asocial type, was depressed following his wife's death four months previously. He made a false declaration to the Public Assistance Committee and, fearing a prosecution, made a determined attempt to gas himself. A 34-year-old man, of poor intellect, had been living alone for several years, following a quarrel with his family. He had been accused of accosting a woman in the street and feared a police charge. He made a slight attempt to cut his throat with a safety razor blade and had possibly also drunk a small amount of liniment. On admission to hospital there was no definite evidence of mental disorder, but he deteriorated rapidly, becoming melancholic and actively suicidal, and it was necessary for him to be certified and transferred to a mental hospital.

A young man, enthusiastic in political activities, prominent in his local circle, fond of the limelight, was dismissed from his work. He felt he could not face his friends, feared to be laughed at, and made an attempt with some effect of dramatization.

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CONDITIONING CAUSES.

Table VIII.

Cause.	Females.	Males.
Solitary, "shut-in" personality	6	19
Erratic, irresponsible personality	8	13
Moral degeneracy	none	4
Feeblemindedness or border-line feeblemindedness	1	5
Emotional instability	11	4
Venereal disease (neuro-syphilis)	none	3
Post-encephalitis	2	none
Epilepsy	1	1
Severe physical ill-health (other)	1	3
Chlorodyne addiction	1	none
Climacteric disturbance	2	..

In a number of cases it was stated that the patient had for years been quiet and unsociable, had few friends or interests, tended to brood and worry and took things much to heart. The prevalence of this type of personality in males was striking. There were 19 cases, and only those were included in this group in which these characteristics were very evident from the history, and were noted by relatives, friends and other informants. In 8 of these cases the attempt was made when the mental state was one of psychosis, and in the majority of these patients it was evident that the mental disorder was a reaction to domestic, economic or physical stress.

Other patients were known to have been impulsive, erratic, unreliable and generally unstable, as was characterized by such facts as quarrelsomeness, cruelty and failure to maintain their dependents, disorderly behaviour in public, alcoholism, consistent failure to hold jobs, previous attempts at suicide and so on.

Four of the male cases had histories of such debauched, immoral and vicious lives that they are here classed as moral degenerates. Especially in these cases it was difficult to obtain a reliable history.

Others, mostly female cases, had shown evidence of emotional instability beyond the normal, and the majority of these had previously been diagnosed and treated as psychoneurotics.

It will be noted that conditioning factors were less frequent in female than in male cases. This is of interest, for in 11 women patients the attempt was the result of domestic stress (7) and love affairs (5), with no evidence of any predisposing factors or abnormal psychological make-up. In the males, however, there was only one case in which domestic trouble was the only ascertainable cause.

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Neuro-syphilis was present in 3 male cases, viz., dementia paralytica, tabo-paresis and tabes dorsalis. The first, a ship's steward, began to have failing sight nine years before. This was progressive, till five years later he had to retire from work. From that time he began to deteriorate mentally and physically, and at the time of admission he was almost completely blind, showed typical physical and biochemical signs of general paralysis of the insane, and was mentally confused, noisy, incoherent and hallucinated. He made a superficial attempt to cut his throat with a carving knife. The second case was clearly one of tabes dorsalis with slowly progressing dementia; he was dull, retarded, depressed, moody and solitary. He also made a feeble attempt to cut his throat with a safety razor blade. The third case, with definite physical signs and a tabetic gold curve, had shown rapid development. He was acutely demented—confused, restless, muttering, agitated and apparently hallucinated. His attempt by hanging was severe, and he was later certified, as was the first case, and sent to mental hospital.

The 2 cases of post-encephalitis epidemica were single girls, æt. 24 and 25. The first showed well-marked Parkinsonianism, had been an inmate of an institution for the past three years, and made her attempt there, by strangulation, during what was apparently a period of mental confusion. She returned to her institution. The second had had an acute attack, definitely diagnosed, during her school years. From that time character changes had been noticeable and persistent, there were frequent domestic quarrels, and after one of them the patient made a slight attempt to cut her throat with a razor blade. She was able to return to her home.

Epilepsy was the basic cause of two attempts, one by a young woman of 27 in a confused state after a seizure, the other by a youth of 23. The latter had been worried by failure to get employment on account of his disability, and made an attempt by hanging, in a state of acute maniacal excitement. Both were later able to return to their homes.

In 4 other cases physical trouble appeared to be the starting-point of the train of circumstances leading to attempted suicide. A married woman of 25 had for some years suffered from very severe anæmia aggravated by two confinements. Her physical state rendered her incapable of coping satisfactorily with her housekeeping. An unsympathetic husband failed to realize her physical disability and treated her with little consideration. There were frequent quarrels, the patient got into financial difficulties, and the husband threatened to leave her. To end it all she made a severe and determined attempt to gas herself. She was discovered in time, and on recovery still wished she had succeeded in her attempt. Prolonged treatment of the anæmia resulted in marked improvement of both her physical and psychological state, and a better understanding by her husband resulted in the patient being able to return home with good hope for future harmony. Physical disorder appeared to be the important causative factor in the development of mental disorder in

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3 men whose attempts were due to their abnormal mental states. Two of these were over 60 years of age, one suffering from carcinoma of the liver with secondary deposits in the lungs, the other from severe debility and marked loss of weight of uncertain cause. Both became depressed, agitated and confused, and one had somatic delusions. Both died. The third was in hospital for the investigation of pain in an old amputation of one foot. He developed persecutory ideas and hallucinations, but quickly recovered normal outlook after his attempt. These three men all attempted to cut their throats, the wound in each case being slight.

An elderly woman; the wife of a tradesman, had been taking chlorodyne for fifteen years. She admitted to drinking a bottle every day. The result was persistent financial difficulty, and the circumstances were such that the economic position would in a few months become hopeless. It was considered that the patient made her attempt, being unable to overcome her habit, as a reasoned solution of the problem and solely for the benefit of her family.

The psychological and physical changes associated with the climacteric appear to have had an influence in only 2 cases in this series. In one, this disturbance associated with domestic troubles seemed to have precipitated a condition of agitated melancholia. In the other, the patient was unable to sustain the stress of physical disorder.

Attitude of the Patient to the Attempt on Initial Recovery.

This was investigated in every case. Of the women, 6 denied any intention, 26 expressed regret or remorse, 4 thought that their action was justified, and 10 others still expressed the wish to die. Four could give no coherent reply.

Of the male patients, 22 expressed regret and realized that their action was a mistaken one, while 4 denied any intention. Eight still had no desire to live and wished they had succeeded, and 3 showed complete indifference. One was uncertain, and 12 were unable to give any coherent expression of opinion.

Observations.

An accurate estimate of the incidence of attempted suicide is at present unobtainable, there being no one authority to whom all cases must be notified and no method of discovering what proportion of cases is reported. The figures given here, however, show that unsuccessful attempts occur much more commonly than successful ones and the relative incidence of the two sexes, namely 6 males to 5 females, is probably correct, at least for this area. It has been noted that up to the age of 25 female cases are very numerous, being twice the total of males. This, no doubt, is due to the hazards of love affairs and of early married life, misfortunes in these circumstances bearing more hardly on the female.

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The reason why attempted suicide is relatively more frequent among females than is suicide is not obvious. Generally the view is held that suicidal attempts by women are commonly of the demonstrative, attention-seeking kind, without real intent to terminate life. There may be justification for this view, but this investigation has shown that women are little less determined in their intention than are men. Impulsiveness, lack of knowledge and preparation result in fewer fatal endings to their attempts.

The patients investigated in this series of cases have been for the most part manual workers, and with few exceptions have been of the small wage-earning class. Whether or not suicidal attempts are less frequent in other grades of social status and affluence is uncertain, because individuals with adequate means can secure private treatment and care, and may be able to conceal the fact of a suicidal attempt. It may be that in the better educated classes, with wider interests and greater compensations for disappointment, suicidal attempts are less common, and when they occur are more often successful.

In the detailed series just over half the males were unemployed, and although unemployment in this area is marked, the number here is far in excess of the average. This might suggest that unemployment is an important factor in attempted suicide, but individual investigation showed that only 6 of the male cases were directly influenced by this circumstance, and even in these cases it was not the sole factor. There is no doubt that prolonged unemployment has far-reaching influences on outlook, health, domestic relations, etc., but the opinion has been formed that the prevalence of unemployment among suicidals is a result of inferiority of personality rather than a cause of suicide. A similar view is expressed by MacEarlean (6) when commenting on the statement that " the rate is nearly twice as high among unemployed as among employed males ".

It is noted, however, that in present times when a minimum economic security is guaranteed for all, the significance of unemployment has to some extent altered.

In the cases under review economic stress has been due, generally speaking, not to lack of adequate income for normal support, but rather to the difficult circumstances that have arisen as a consequence of unwise expenditure—borrowing, gambling and the like—and where necessary repayment was urgent or exposure threatened. In most of the female cases the influence of such a state of affairs on the emotional relationship between the patient and others was the main reason for the suicidal attempt.

Table V shows that nearly 11% of 656 admissions during 4 years were certified and transferred to mental hospitals. This percentage does not indicate the number who were mentally disordered at the time of their suicidal attempts. It has, however, some prognostic significance, although it must be noted that the duration of retention in observation wards is not equal in

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all cases. Acute cases, considered recoverable, are retained as long as possible, chronic cases who have become quiescent and no longer actively suicidal are often claimed by relatives, and vacancies in mental hospitals are not always available.

The detailed investigation shows that 32% were psychotic on admission, but only 13% (9 males and 4 females) were certified. This would suggest, if the corollary holds true, that probably 27% of the 4-year total were psychotic on admission. It might then be reasonably assumed that about a quarter of the cases of attempted suicide in this area are of unsound mind at the time of their attempts. Lendrum (Detroit) (7), investigating 1,000 cases of attempted suicide, states that 23% received some sort of psychiatric diagnosis but only 1·9% were committed to mental hospitals. Stearns (Massachusetts) (8) states that one-third of his series appeared to be frankly insane, while in this country Norwood East (9) found that 14·1% of 1,000 cases of attempted suicide were due to insanity. The last series, however, was a selected group in that they were males concerning whom police action had been taken. In the first and last of these series alcoholism was cited as one of the chief causes of attempts, but in the present investigation in all but 2 cases this factor was merely contributory.

It might seem, on consideration of the data of this investigation, that the adverse circumstances to which the patients had been subjected were exceptional. The results obtained in these cases, however, were very similar to those noted over a prolonged period. Generally, the stresses prompting the suicidal attempts were no more acute or severe than those to which the generality of people are required to adjust. The reason for the reaction by suicidal attempts in these cases lies in the personalities of the individuals, and furthermore, many of the difficulties with which these patients had to contend were the product of abnormality in themselves. The proportion of individuals who showed characteristics of personality deviating from the normal (especially among the males) was striking. In Table VIII there has been no attempt to classify on any scientific basis, but merely to note personal characteristics which have influenced behaviour, and which have been elicited by observation of the patient, and gathered from histories and the evidence of associates. Fairbank (10) has stated that the only outstanding common characteristic found in a study of personality traits in suicidals, and noted in about one-third of his cases, was the so-called "rigid" personality as described by Wendell Muncie (11). In this series the prevalence of the introverted, solitary and asocial type was notable. Such individuals tend, under certain circumstances, to arrive at an altered sense of values and to form unreasonable judgments. The attempts of the erratic, undisciplined type are easy to understand, and possibly one reason why so many are found among attempted suicidals is because they fail in their attempts at self-destruction.

A number of cases, mostly females, had shown evidence of emotional

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instability beyond the normal, and several had previously been treated as psychoneurotics. Gordon (12) expresses the view that psychoneurotics do not commit suicide, and this view is supported by Brend (13), who cites the extreme rarity of suicide amongst the large numbers of psychoneurotic patients treated in Ministry of Pensions Hospitals. Such has also been my experience, and it would appear that the evidence of this is so strong that in suicidal attempts by psychoneurotics, intention of self-destruction should be doubted or the diagnosis of psychoneurosis questioned.

Whether attempted suicide should be viewed and treated as a medico-psychological problem or from the legal aspect as a misdemeanour is still an open question (14, 15). This investigation has shown that the great majority are in need of treatment and come within the province of medicine. A small proportion, the amoral, self-seeking types, require disciplinary action. In this connection it is noted that the attitude of the police is sympathetic and open-minded. They willingly accept medical opinion ; the decision as to legal procedure and the actions taken are based on the nature and possibilities of each individual case. This is as it should be.

The influence of hospital care presents several points of interest. In the psychotic cases efforts are directed to the mental disorder, the suicidal tendency being secondary to this. Other cases whose attempts have been precipitated by domestic, economic and other environmental stresses as a rule quickly discard their suicidal outlook. It would appear as if their action had released an unbearable pressure. Michael and Grimes (16) and Schneider (17) have commented on this rapid change of attitude after impulsive attempts, and the latter states, further, that the patient who waits patiently until he is discharged in order to make a fresh attempt appears to be extremely rare. Such has been our experience. A large proportion of suicidals would appear to have kept secret their fears, dwelt constantly on their difficulties, been unable, on account of increasing emotional bias, to maintain a true perspective, and made the suicidal attempt as an escape from what seems to them an intolerable situation.

In hospital, under conditions sheltered from ordinary life they can take a more objective view. They are enabled to discuss and disentangle their mental complexities, and there is an opportunity for readjustment with relatives and associates. Frank discussion of his attempt with the suicidal, so often avoided for reasons of delicacy or fear of harm, is clearly indicated. The subject is uppermost in the patient's mind, and avoidance of it will hinder the return of a wholly healthy outlook. Bond (14), commenting on this unwillingness to remind a patient of his suicidal attempt, states very truly that the return to " accessibility " is " one of the particularly favourable moments for vigorous psychotherapy ".

In the demonstrative cases the act has served its purpose and the patient has satisfied his or her need.

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Many patients on discharge from hospital to their homes still require psychological treatment or supervision as out-patients. Although their internal tension may have been satisfactorily relieved or their present difficulties cleared away, not a few, by reason of inferior stamina and resistance, will continue to require help on resuming their ordinary lives. The psychological clinic, in the simplest form of its service, will provide them with necessary "moral support". Such a clinic, however, will have its strongest appeal at present when divorced from mental ward or mental hospital.

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Part II.—Reviews.

A Preface to Nervous Disease. By STANLEY COBB, M.D. London: Baillière, Tindall & Cox, 1936. Pp. xi + 170. Price 13*s.* 6*d.*

The writer warns us that he is only dealing with those anatomical structures the physiology of which is known, and discussing only physiological processes for which there is at least a fairly well-established anatomical correlation.

He points out wisely that the autonomic nervous system and the central nervous system have developed side by side; neither may be regarded as older than the other. His chapters are devoted to the autonomic nervous system, the segmental and suprasegmental aspects of the cerebro-spinal nervous system, motor integration and locomotion, functional localization in the cerebral cortex, consciousness and the mind-body problem, cerebral circulation, cerebro-spinal fluid, paths of infection to the nervous system, general histological pathology, the peripheral nerve and neuritis, special histological pathology, and epilepsy and the psychoses. It is pleasing to see stress placed on the absurdity of the upper and lower neurone conception. He comments on the fact that lizards, which are extremely active creatures with most complex motor functions, have no upper motor neurone at all! A combination of simply upper and lower motor neurones would be a very uneven, inco-ordinated mechanism.

The author shows the importance of the Gestalt theory in discussing cerebral function—the brain acts as a whole in many ways. He would have us believe that Sherrington's integrative action of the nervous system supplies a working hypothesis on which we may build a superstructure which will explain the working of the higher mental processes. He does not see much difference between the mental and non-mental functions of the nervous system, whether these are studied by chemical, physical or microscopical methods.

The old-fashioned division of disease into the two categories of functional and organic comes in for some rough handling. The author wisely indicates that the deciding factor is simply the instrument used. Twenty-five years ago paralysis agitans was considered a functional neurosis.

The trend of recent work is to support the hypothesis that the cerebro-spinal fluid is a dialysate. If the choroid plexus secretes the fluid then the mechanism is similar to that of the kidney, as for one thing secretion granules do not occur in the ependymal cells.

The author draws attention to the recent work of Morrison, who maintains that the function of the oligodendroglia cells is to deposit myelin around the axones during the process of myelinization in early life: possibly these cells may also help in the process of repair by depositing myelin on axones stripped of their sheaths by demyelinization.

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The writer falls into the common error of using a hyphen in " Argyll Robertson ".

He does not regard epilepsy as a disease, and points out that the important processes in most fits are altered conditions of the nerve impulses, increased irritability of the nerve-cells, oxygen lack, and changes in the acid-base equilibrium.

The illustrations are very good, and we can thoroughly recommend this book to any who wish to understand the simpler workings of the nervous system. G. W. T. H. FLEMING.

Essentials of Psychopathology. By G. H. HENRY. London: Baillière, Tindall & Cox, 1935. Pp. x + 312.

An excellent book that provides a good all-round introduction to the subject. " Psychopathology " is here used with a somewhat wider connotation than usual, and covers most of the ground that might be included under some such heading as " mental symptoms and their causation ".

Throughout the book there is a nice balance between the psychogenic and the physiogenic points of view, and the author successfully shows how different interpretations may depend more upon the mode of presentation of the facts than on the facts themselves. Thus in Chapter I, on heredity and environment, he details a patient's family history on the usual lines of grandparents, uncles, aunts, etc., and from it he draws certain obvious conclusions concerning the importance of hereditary factors. He then proceeds, however, to recount the same history in terms of the relationships between the patient and her relatives and of the patient's attitude and reactions to her surrounding difficulties; the whole picture now takes on a different aspect, and personal psychogenic factors come into prominence while hereditary influences seem to recede into the background.

The chapters on personality and constitution give a very good survey of modern research into physical and mental constitution in mental disease. (including the work of Lewis, Kretschmer and Stockard) and a fair account of the typologies of Jung, Bleuler, Adler and others, though in the description of Jung's theories accuracy has had to give way slightly to condensation. After a summary of cerebral functions comes a chapter on toxic influences, in which the author adopts a broad point of view that allows well for the intermingling of mental and physical factors in the production of psychoses. The theme is further developed in the section on relation of disease to personality disorders, and is then followed by an account of vegetative functions and emotional reactions.

Two important chapters are those on personality integration and mental dynamisms. Both are remarkably adequate in spite of their brevity. In the former a general account of mental development is given, largely along psychoanalytical lines, though the terminology is not unduly technical and the approach broad. The not unusual error of identifying the " super-ego " with a conscious conscience is made, but apart from this and other minor faults the account is clear and pleasantly descriptive. Mental dynamisms are dealt with under eleven sub-headings (identification, introjection, condensation, etc.), and are all the better understood for the insertion of thumb-nail sketches of individual cases.

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In the long chapter on childhood disorders we are given the benefit of a detailed account of a case, including verbatim reports of the patient's utterances on successive dates. The exposition is rendered of greater value by the preliminary grouping of the patient's remarks in respect of the topics they touch upon and the trends they disclose. Thus one section of statements is headed "Habits of eating", another "Complaints"; others again are "General anxiety", "Sexual references", "Desire for Baby", "Ideas of Death". The next two chapters deal with case-records and methods of examination respectively, while a general account of psychopathological phenomena concludes this attractive volume.

No doubt a book of this kind cannot satisfy everybody, but we have here a well-balanced and sober account that steers clear of extremes, one which can be heartily recommended, especially to those who are approaching the subject as novices. The bibliography is not very full (perhaps a "good" fault), and the index is short of several words (such as "ritual", "homosexual", "histamine"), all of which figure fairly prominently in the text.

J. ERNEST NICOLE.

Biological Time. By LECOMTE DU NOÜY. London: Methuen & Co., pp. 180. Price 7*s.* 6*d.*

The purport of this book may be stated in a few words. Everyone knows how time seems to go faster as one grows older. The author's thesis is that this subjective impression, far from being trivial, is really perfectly correct, since he maintains that there is a biological time which flows at a different rate from physical time.

The book is divided into three parts. The first is an introduction and deals with technical methods. The second part gives the facts on which he bases his theory. The facts are drawn mostly from the data of the rate of cicatrization of wounds and the rate of growth in tissue cultures.

It is known from the work of the author and Carrel that in the healing of a superficial wound the surface is approximately an exponential function of the time. That is to say:

$$S = S_{\prime\prime}\, e^{-kt}.$$

It was found that the constant k was a function of the patient's age. Now multiplying t by k in the equation above is really equivalent to changing the time scale. This means that it is permissible, from the logical-mathematical point of view, to regard physical time (e.g., the duration of a swing of a pendulum) as progressing at different rates for the old and the young. Thus if a young man heals a wound while a given pendulum swings 100 times, and an old man heals a similar wound while the pendulum swings 200 times, then, if we take the swing of the pendulum as our standard time, we may say that the old man's wound took just twice as long to heal; or we may take the healing of the wound as our standard time and we may say that the pendulum swings twice as fast for the old man, since it performed twice as many swings in the standard time.

This, briefly, is du Noüy's argument. He then discusses the nature of time so as to see whether his point of view is really incompatible with the usual point of view. He decides that it is perfectly permissible to assume

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that there is an "inner", biological time as well as an "outer", physical time. His main argument here is that our whole experience of time is intimately linked up with psychological, as well as with physical, factors. Thus, if we human beings had no memory, we would be fundamentally incapable of forming any concept of time: we would simply live in an eternal present. Thus the concept of time brings in that of memory, just as any concept of memory must bring in that of time. Unfortunately, all discussions of the nature of time involve difficulties of very high order, and his argument may or may not be convincing.

But the real test of all hypotheses is to see how they develop and what they lead on to. The author seems to have little to add in the way of further deductions. But unless the hypothesis is quickly developed further it is likely to join the legion of interesting, but forgotten, hypotheses.

W. R. ASHBY.

A Quantitative Analysis of the Structure of the Cerebral Cortex. S. T. BOK. (*Kon. Akad. Wet., Verhand.* [Tweede Sectie], Dl. xxxv, No. 2, pp. 1–55, 1936.)

This monograph deals with measurements of the cyto-architectonic features of the human cerebral cortex.

Dr. Bok has made measurements of all the nerve-cells in one strip of a section from the *area temporalis superior posterior*, using one of Economo's sections. He measured 1,546 cells in all, estimating the nuclear volume and depth of each cell from the pia mater. With these measurements he has attempted to find some simple rule for specifying the cyto-architectonic nature of a given piece of cortex.

He is of the opinion that a complete description of cell size and its distribution can be given, for any particular piece of cortex, by four numbers.

Unfortunately, the validity of his conclusions depends on whether he has chosen the right functional forms for the regression lines in his data. The figures for nuclear volume show a wide scatter when plotted against depth from the pia mater. Under such conditions it is possible to get a variety of functions (parabolic, logarithmic, etc.) to fit reasonably well, especially as the regression lines are almost, if not quite, straight. In such a case it is vitally important to use the best possible methods for testing "goodness of fit". The author's method is to draw the diagram and to leave it to visual impressions to be the judge. Such a method is known to be unreliable. It is a pity that the really accurate arithmetical methods were not used. There is no mention of the "method of least squares" or of the "analysis of variance".

But the real test of the author's work will come when he makes similar measurements on other regions of the cortex—preferably of widely differing types. If it is found that his four numbers will still describe completely the distribution of cell size and density of cells, then he will have achieved a really great piece of work. It seems to be almost certain that there must be some real simplicity behind the apparent complexity of the cyto-architectonics of the cerebral cortex. Bok's work, which is at present incomplete, is certainly an ingenious and determined attempt to find this simplicity.

W. R. ASHBY.

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Erbanlage und Epilepsie. (Hereditary Disposition and Epilepsy.) By K. CONRAD.

I. "Untersuchungen an einer Serie von 253 Zwillingspaaren," *Zeitschr. f. d. ges. Neur. u. Psychiat.*, cliii, p. 165, 1935.

II. "Die konkordanten Eineiigen," *ibid.*, clv, p. 254, 1936.

III. "Die diskordanten Eineiigen," *ibid.*, clv, p. 509, 1936.

To illustrate the present position of the problem of heredity in epilepsy a few facts may be mentioned which throw a characteristic light on the state of affairs. At the International Neurological Congress in 1935 epilepsy was one of the principal topics; 50 papers dealt with it, of which only two dealt with the problem of heredity. Previously, in 1932, at a French neurological meeting, Abadie, the first and certainly one of the most prominent speakers of the general discussion on epilepsy, had refuted the hereditary character of epilepsy. He contrasted his view with the opinions of former French authors (such as Grasset and Rouzier), who stated at the end of the last century that "epilepsy is the most frequent manifestation of heredity in medicine". To-day, according to Abadie, "epilepsy can no longer be regarded as a hereditary disease, or as a constitutional illness which is transformed from one generation to the other, in more or less identical form". One of the two papers mentioned above was by Kurt Conrad, who disclosed here the first results of his investigations; these have now been completed and published in three papers, a detailed record of which may be of some interest.

The study of twins, used by Conrad, is certainly the most exact method in human heredity research. It is founded on Galton's ideas, and has since been developed into a sensitive instrument, especially with regard to the diagnosis of uni-ovularity. The idea is that uni-ovular twins have an identical genic equipment; binovular twins are no more alike than ordinary sibs. If there is a high concordance (= equality in phenotype) in uniovular twins, as regards a particular character, while the binovular twins are not much alike, one would suppose the character to be largely hereditary. Environmental factors are supposed, as a rule, to have an equal influence on binovular and uni-ovular twins. So, if the uni-ovulars are *not* much more concordant than the binovulars, environmental factors can be assumed to be decisive.

There have been investigations of epileptic twin series before Conrad, though he does not consider them of high value. A paper by Schulte deserves mention, however, He applied the method to a series of 24 twins, collected from the material of the Berlin Clinic. He found 10 uniovulars, of whom only two were concordant with regard to epilepsy. These results are of special importance because of the entirely different material from which they were obtained. Whereas Conrad took institutional patients only, Schulte worked with slighter forms of epilepsy attending an out-patient department.

Although Schulte satisfied most demands of modern research methods, Conrad objects to his procedure in *collecting* the twin propositi. They do not form an unselected representative material. That is proved—as Conrad thinks—by the small number of pairs, of whom one twin died in infancy. This figure remains below the average of the general population. Furthermore the number of Schulte's uniovulars is too high in comparison with the average occurrence, and finally his total number of twins seems rather small.

In order to avoid these and other errors of former investigators, Conrad collected the details of all epileptics in German mental hospitals or similar

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institutions at a certain date (February 1, 1934) and made inquiry at the appropriate registration office as to which of these had been one of twins. Among the 12,561 persons inquired about were 258 twin-born, of whom 83 were of the same sex and lived beyond the fifth year. Only twins of the same sex can possibly be uniovulars. Of these 83 the author was able personally to examine 63. In the remaining 20 cases the diagnosis of uniovularity could be ascertained with the help of photographs or descriptions. Thus he finally found 30 uniovular twins, which really constitute his crucial material, and are contrasted with 127 binovulars, of whom both twins lived beyond the 5th year.

The first and most interesting result of this collection was the finding of a greater frequency of twin births in epilepsies than in the average population. The obvious explanation that the difficult labour associated with a twin birth can cause epilepsy through traumatic cerebral lesions is repudiated by Conrad. He finds that the increased number of twins was entirely confined to the binovulars in his material. The number of uniovular twins was below average.

Birth trauma one would expect to effect uniovular and binovular twins equally. That in his material there was only an increase in the binovulars must—according to Conrad—be explained otherwise.

Furthermore, as regards position in the family, the number of first-born among his twins was not above average; on the contrary, first and early births were below statistical expectation.

As regard the order within the twin birth, Conrad found equal numbers of first- and second-born among his epileptics. One would expect the first-born to be more particularly affected by a difficult labour. Therefore this finding also speaks against the influence of birth trauma.

The result is in striking contrast to the findings of other authors, e.g., to those of Schulte, who claimed to prove by X-ray examination of the skull the effect of the more difficult labour on the first-born twin.

Looking for an explanation of the increased twin frequency, Conrad assumes a "biological correlation" between epilepsy and tendency to twin birth. An inclination towards poly-ovulation, especially in middle-aged women, is, he thinks, correlated to the epilepsy-predisposition, as has been found in other morbid conditions, viz., myoclonus epilepsy, Huntington's chorea and mental deficiency by other authors. It is thus regarded as a kind of stigma of degeneration. Conrad tries to confirm this view by proving an increased twin frequency among the brothers and sisters of his twin propositi.

The most important preliminary question of all twin investigations is the definition of what shall be called concordant. This is often difficult to establish when one deals, e.g., with an illness which manifests itself in various symptoms. It is relatively easy in epilepsy, the epileptic fit being the main and unambiguous symptom. Conrad also regards certain definitely observed equivalents as of equal value, viz., absences with loss of consciousness and twilight states. He excluded spasmophilia and other transient convulsions in infancy from concordance. This is one reason why he left out from his calculation the 94 cases of whom one twin died in early childhood, often following convulsions. Moreover, the diagnosis of ovularity was, of course, impossible in these cases. Finally, he stresses the point that mental deficiency and other neurological or psychological anomalies should not be regarded as concordant as long as they are not accompanied by epileptic fits.

The difference in concordance between uniovulars and binovulars that he found by this method is striking: among the 30 uniovular twins, there were 20 of whom both were epileptic; in 18 cases both had seizures, in 2 cases one

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seizures and the other equivalents; whereas among 127 binovulars only 4 pairs were found in whom both were epileptic. Expressed otherwise, *the percentage of concordance in monovulars was* 66·6, *in binovulars* 3·15%. Even if one were to include mental deficiency and psychopathy into the calculation, it would make little difference.

The next step in Conrad's investigation is concerned with the much discussed problems of *idiopathic epilepsy*. There was and is a general inclination towards narrowing the concept of idiopathic epilepsy. Numerous authors have declared it as something provisional, which will disappear the moment the cause of all epilepsies is revealed. The epileptic fit is generally recognized as a universal response of the brain to any sufficiently grave stimulus and may occur in any person. Artificial epilepsy produced by camphor injections has been used as a treatment of schizophrenia, and the recent treatment of the same illness by insulin shock also evokes epileptic seizures in many cases, quite independent of any predisposition.

In contrast to these theories and observations, Conrad tries to separate clinically the idiopathic from the symptomatic epilepsies in his material. For the former he takes all propositi without apparent causation of the disease; for the latter he collected all cases in which there were neurological symptoms, manifest deformities like hydrocephalus, etc., and recognizable causes like trauma, lues, encephalitis. If one compares the number of concordant cases within the two groups, the percentage of the former is found to be increased in the uniovulars and unchanged in the binovular twins. Thus the existence of a group of purely genic idiopathic epilepsy seems established, and Conrad denies the possibility of entirely resolving it into the symptomatic forms.

He concludes the statistical part of his investigation by working out the probability of manifestation of the epileptic "*Anlage*". He calculates that, disregarding any distinction into idiopathic and symptomatic, persons having the epileptic predisposition will actually develop epilepsy in 80% of cases.

Two more papers of Conrad exhibit the main clinical material, on which the statistics of the first paper is founded. There one finds the measurements and observations on which the diagnosis of uni-ovularity was based, furthermore photographs, case-histories and clinical findings in all his twins. After the doubtful cases and the propositi who had to be counted twice for statistical reasons were excluded, this material is reduced to 22 pairs of patients. Of these Conrad regards 10 as "absolutely concordant", 5 as "relatively concordant" and 7 as discordant.

Among the 10 absolute concordants one finds several twins of whom it would be difficult to imagine a closer identity of symptoms, either in form or in temporal order. In others there was some difference with regard to the later development and course of the illness, but the fits began in each pair at the same time of life, and the symptoms are very similar throughout.

The difference between these patients and the 5 "relatively concordant" twins is only a matter of degree. There were also epileptic fits or at least equivalents in both twins, but the time of their appearance, their duration and the other signs of the illness are more or less different. There is for instance one pair, now æt. 31, of whom one twin has had only one epileptic fit up to now, but the other has shown a chronic progressive epilepsy. Numerous neurological, psychological and endocrinological complications were present in most cases, which make a clear-cut clinical diagnosis rather difficult.

Such a diagnosis is relatively easy in the non-concordant pairs; here the

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causation of the epilepsy in the propositus twin is clearly to be seen. There are 3 cases of post-traumatic epilepsy, several after encephalitis or due to similar gross factors.

However it is just the discordant uni-ovular twins which might be of special interest and convey novel information. Since uni-ovulars carry identical genic equipment, there is nothing astonishing in their being equal in the phenotype provided the environmental influences are identical; the latter are so in most of Conrad's twins. In discordant cases one has a special opportunity to learn about the factors which favour or hinder manifestation of the epileptic predisposition. Here the true medical interest comes in: one would like to know how to avoid the manifestation in the remaining 20% by prophylactic means. Conrad deals with this problem in most of his cases in great detail; but he does not arrive at anything really new. He certainly sees all the complications and seeks for solution as far as possible.

The final conclusions he draws from the result of his research are neither one-sided nor exaggerated. He repeatedly emphasizes that the choice of his material, containing only institutional cases, restricts the value of his results with regard to the sum total of epilepsies. He does not think that he has proved the genetic nature of all epilepsies; but he recognizes—if I understand him rightly—a sliding scale from predominantly genetic to entirely environmental cases. He is opposed to those authors who seek to eliminate predisposition as causing epilepsy, and thinks the term "idiopathic epilepsy" justified for a central group of patients who are destined to become epileptics thanks to their genetic organization. How large this central group is within the total number of epilepsies he is unable to say. But the establishment of the hereditary origin of epilepsy in a certain number of cases is of great importance, especially if these patients coincide with those clinically diagnosed as idiopathic.

W. MAYER-GROSS.

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Part III.—Epitome of Current Literature.*

1. Anatomy and Physiology.

Effect of the Injury of the Lobulus Posterior Medianus (Ingvar) of the Cerebellum on the Blood-pressure, the Blood-sugar Content and the Adrenaline Secretion in Non-anæsthetized, Non-fastened Dogs. (*Tôhoku Journ. Exper. Med., vol. xxvii, pp.* 218–44, 1935.) *Wada, M., Seo, M., and Abe, K.*

The lobulus posterior medianus of the cerebellum was punctured in 8 non-anæsthetized dogs. The quantity of adrenaline in the suprarenal vein blood (collected without fastening the animal or causing any excitement) was determined by the method of the rabbit-intestine segment and the paradoxical pupil reaction of the cat. The operation resulted in an increased rate of adrenaline secretion (0·00015–0·00035 mgrm./kgrm. body-weight/min. above the normal for both glands), reaching a maximum after 5–15 minutes and continuing for 1–2 hours. The total amount secreted for 2 hours after the operation equalled 0·012 mgrm./kgrm. body-weight. The hyperglycæmia was very slight. The heart-rate increased somewhat in most cases and never decreased. When the puncture was made posterior to the second fissure there was a marked and continuous increase in arterial blood-pressure, while puncturing anterior to the fissure produced a smaller and transitory hypertension. The former puncture invariably produced nystagmus.

S. A. Corson (Chem. Abstr.).

Effects of Urethane on the Sigmoid Area of the Cortex and Experimental Urethane Catatonia. (*Riv. Patol. Sper., vol. xiv, pp.* 101–20, 1935.) *Mondio, E.*

Urethane (25%) applied directly to the sigmoid area of dogs produces depression of reflex activity and of excitability of these centres, but does not produce variations in the tendency to epilepsy from afferent stimuli. Subcutaneous injection of 300–500 mgrm. of urethane, however, produces symptoms of catalepsy, without modifying the activity or excitability of the sigmoid centres.

E. Bame (Chem. Abstr.).

Effect of Pituitrin on Conditioned Reflexes. (*Arch. Sci. Biol.* [*U.S.S.R.*], *vol. xxxvii, p.* 111, 1935.) *Asratyan, E.*

The effect of subcutaneous injection of pituitrin on conditioned reflexes (salivation) in the dog was studied. Doses of 1–1·5 c.c. diminished the reflex considerably in 40–90 minutes. Doses of 2–2·5 c.c. abolished positive conditioned reflexes completely in the same time. Doses of less than 1 c.c. had a weakening effect. In all these cases unconditioned reflexes were hardly affected. Very small doses on investigation after a shorter interval found unconditioned reflexes increased at first, then all reflexes abolished. E. Bame (Chem. Abstr.).

Effect of Adrenaline, Anæmia and Carbon Dioxide on the Vasomotor Centre. (*Arch. Intern. Pharmacodynamie, vol. li, pp.* 463–87, 1935.) *Nowak, J. G., and Samaan, A.*

Adrenaline exerts no direct central vasodilator effect. The persistence of a slight vasodilator effect after carotid sinus denervation and depressor-nerve section,

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in both perfused isolated leg and perfused isolated head experiments, is explained on the basis of sudden changes in perfusion of the vasomotor centre by the adrenaline hypertension. The vasodilator reactions obtained after adrenaline injection were reproduced in the isolated perfused head experiments by mechanically increasing the perfusing pressure. Well-marked curtailing of the cephalic circulation in dogs deprived of the four buffer nerves (namely, the cardio-aortic and carotid sinus nerves) has no effect on the vasomotor centre. When, however, the anæmia is extreme, an abrupt increase of the vasomotor tone is observed. Perfusion of the head, which is connected to its trunk by the spinal cord only, with hyperapnœic blood is associated with marked vasomotor stimulation. This effect is partly reflex, owing to afferent impulses from the "chemical receptors" of the carotid sinuses. Other points concerning the central and peripheral effects of carbon dioxide are discussed. E. BAME (Chem. Abstr.).

The Mineral Composition of the Blood of a Dog Deprived of One Cerebral Hemisphere and of the Motor Cortex of the Other Hemisphere. (*Arch. Sci. Biol.* [*U.S.S.R.*], *vol. xxxviii, pp.* 403–6, 1935.) *Abuladze, K. C., et al.*

No significant differences were found in regard to whole blood chlorine and serum phosphorus, sodium, potassium, calcium, magnesium and sugar between the blood of one operated animal and that of control dogs.

W. A. PERLZWEIG (Chem. Abstr.).

Effect of Environmental Temperature on the Blood-sugar Content of the Heat-punctured Rabbit. (*Tôhoku Journ. Exper. Med., vol. xxvii, pp.* 473–86, 1935.) *Ichikawa, K.*

The skull-bone of non-anæsthetized, non-fastened rabbits was perforated and the dura mater opened one day before the puncture of the corpus striatum. The body temperature rose to 41°–41·7°. Only a slight transitory hyperglycæmia (determined according to Hagedorn and Jensen in blood drawn from the marginal vein of the previously denervated ear) occurred in some of the rabbits. When the punctured and control animals were heated, the body temperature in the control rabbits rose to 40°–41° (temperature of surroundings was 34°–39°), and a slight hyperglycæmia occurred in some. The body temperature of the punctured rabbits rose to 43°–44·5° and the blood sugar increased, reaching a maximum (0·14–0·28%) after 2–4 hours (1–2 hours after the body-temperature maximum).

S. A. CORSON (Chem. Abstr.).

Presence and Distribution of Reducing Substances in Nerve Substance. (*Atti Accad. Lincei, vol. xxii, pp.* 359–65, 1935.) *Mitolo, M.*

Different parts of the nervous system of the frog, white rat, pigeon, guinea-pig, rabbit, cat, sheep, horse and bull, such as the spinal marrow, optic lobes, brain and cerebral hemispheres were examined for their power of decolorizing the indicator 2, 6-dichlorophenolindophenol. A. W. CONTIERI (Chem. Abstr.).

The Hypothalamus and Cardio-vascular Reactions. (*Arch. Intern. Pharmacodynamie, vol. liii, pp.* 137–76, 1936.) *Van Bogaert, A.*

Electrical and chemical stimulation of various regions of the hypothalamus of dogs causes either a rise or fall of blood pressure, and changes in the cardiac rhythm which are independent of the blood pressure changes. The region causing a rise of blood pressure is anatomically distinct from that causing a fall. Accompanying the rise of blood pressure is a hyperglycæmia dependent on the presence of the adrenals and a lowering of the blood lipides and potassium, and a rise of blood calcium. Because of the strength of the stimulus needed to produce effects it is concluded that the hypothalamus does not normally originate impulses, but acts as a relay for impulses originating in other parts of the central nervous system.

M. L. C. BERNHEIM (Chem. Abstr.).

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The Nerve Centres Controlling Adrenaline Secretion. The Presence of Centres in the High Cervical Region of the Dog. (*Arch. Intern. Pharmacodynamie, vol. liii, pp.* 71–80, 1936.) *Jourdan, F.*

By the rise of blood-pressure caused by anæmia of the high cervical region it is concluded that this contains centres which cause the liberation of adrenaline from the adrenals. M. L. C. BERNHEIM (Chem. Abstr.).

Cerebral Control of Fat Metabolism. (*Orvosi Hetilap., vol. lxxx, pp.* 336–38, 1936.) *Molnar, István, and Marsovszky, Pál, jun.*

Experiments with hares showed that an increase of cerebral pressure diminished the fat content of liver, kidneys and musculature. The increase of cerebral pressure was effected by intracisternal injections of kaolin suspensions. The observed changes in fat content were not caused by insufficient nutrition.

S. S. DE FINÁLY (Chem. Abstr.).

2. Psychology and Psychopathology.

Inhibitions, Symptoms and Anxiety (contd.). (*Psycho-Anal. Quart., vol. v, p.* 261, *April*, 1936.) *Freud, S.*

Continuing his investigations into the relationship between anxiety and phobia formation, Freud finds that the anxiety in these cases is an affective reaction of the ego to danger, the danger which is here warned against being that of castration. By means of the phobia one external danger is merely substituted for another, the ego escaping anxiety by the process of avoidance or inhibition. It is noted that there is no difference between this anxiety and the reality fear normally manifested by the ego in situations of danger, other than the fact that the content of the former remains unconscious and enters consciousness only in distorted form.

In the case of the compulsion neuroses the motive force behind the symptom is the ego's fear of its super-ego. The hostility of the super-ego is the danger situation that must be avoided—again an extension of the punishment of castration. For just as the super-ego is the father become impersonalized, so the dread of castration, which he threatens, has become converted into definite social anxiety or dread of conscience.

The traumatic neuroses, so often the sequel to exposure to danger to life, are a result of death anxiety. But nothing similar to death has ever been experienced, and therefore the fear of death is to be regarded as an analogue of the fear of castration. The situation to which the ego reacts is the state of being deserted by the protecting super-ego, by the powers of destiny, which puts an end to security against every danger.

It is concluded that anxiety is a specific state of unpleasure accompanied by motor discharge along a definite pathway. Anxiety originally arose as a response to a situation of danger, and is regularly reproduced whenever such a situation recurs. Two possibilities with regard to its appearance may be distinguished: the one, inappropriate and inexpedient, in response to a new situation of danger; the other, a useful one, as a means of giving warning of and averting such a situation. In every case the danger situation, giving rise to the anxiety state, is found to be one of privation—a reaction to the perception of the absence of the object. Ultimately the most basic anxiety of all, the "primal anxiety" of birth, arises in connection with separation from the mother.

The next transformation, castration anxiety, which makes its appearance at the phallic phase, is a separation anxiety also, and is similarly conditioned. The danger here is a separation from the genital. The point of connection with the earlier content of the danger situation is the highly narcissistic value attaching to the penis, the possession of this organ guaranteeing a reunion with the mother or mother-substitute in the act of coitus (Ferenczi). Finally with the impersonalization of parental authority at whose hands castration was feared, the danger becomes

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more indefinite. Fear of castration develops into dread of conscience—into social anxiety. That is, it is the anger, the punishment of the super-ego, the loss of its love, which the ego apprehends as a danger, and to which it responds with the signal of anxiety. This final transformation occurs in the latency period.

All these danger situations and anxiety determinants may persist alongside one another, causing the ego to react with anxiety at a later period than the appropriate one; or several of these may become operative simultaneously. Possibly there also exists a close relationship between the danger situation which is effective in the given case, and the form of neurosis which develops.

STANLEY M. COLEMAN.

Dreams and Dream Interpretation of the Diegueño Indians of Southern California. (*Psycho-Anal. Quart., vol. v, p. 195, April,* 1936.) *Toffelmier, G., and Luomala, K.*

This study furnishes an example of a primitive people to whom dreams and their interpretation constitute a vital part of the culture pattern. The investigation is assigned to rigidly trained dream doctors, whose speciality it is to interpret dreams, and to use them as a probe into the problems of their neurotic patients. The Diegueño recognize the existence of functional mental disorders, the dreams revealing to the dream doctor the patient's conflicts and desires, which are usually of a sexual nature. A system of therapy has been developed according to which sexual hysterias are classified and treated. It is a claim of the dream doctor that although, owing to his magic power, he knows his patient's dreams before he hears them, therapeutic value lies in the narration of the dreams by the patient, and in the free communication between doctor and patient. These dreams are, for the most part, direct sexual dreams with little symbolism. It is found that the affect of an anxiety dream varies with the tribal status of the dreamer, and becomes exaggerated when that status, social or sexual, is menaced.

STANLEY M. COLEMAN.

Exhibitionism and Exhibitionists. (*Int. Journ. of Psycho-Anal., vol. xvii, p. 321, July,* 1936.) *Christoffel, H.*

In exhibitionism, from the point of view of the erotic instinct the effect desired is the furtherance of his suit; from that of the death-instinct it is to inspire terror. While exhibitionism presupposes the presence of a second person, it is essentially an act which has reference to the subject himself; the narcissistic, passive element prevails over that of active object libido. Probably one of the earliest forms of exhibitionism is the screaming fit of the infant, the aim being to obtain special attention. Again exhibitionism is one of the factors in the complex phenomena of stammering.

Male genital exhibitionism is a regressive post-pubertal phenomenon. In the child's polymorphous instinctual activity exhibitionism need not have been primarily prominent; it is mobilized secondarily, at the time when, normally, mature genitality should be asserting itself. The hetero-sexual object relation is retained, at all events manifestly; phallicism is retained, but it becomes essentially passive. The essence of the catastrophe is that, in his relation to adult women, the exhibitionist reverts to the situation of the suckling—his relation to a woman in his earliest days. The main feature therefore is the activation of oral incorporation. His oral sadism is projected on to the woman, so that she takes on the significance of a being who incorporates by devouring. By a process of displacement another element of his orality comes into play in his scopophilia: the sexual object is, as it were, devoured with the eyes. Constitutionally this orality is not particularly strong, but its elaboration is defective as a factor in ego-development. Invariably the exhibitionist is an intelligent person, whose thirst for knowledge and craving for recognition has never been fully satisfied. Ultimately it is the father whom he reproaches for this.

STANLEY M. COLEMAN.

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3. Psychiatry.

Suicide and Mental Disease. (*Arch. of Neur. and Psychiat., vol. xxxvi, p. 1, July,* 1936.) *Jameison, G. R.*

The unconscious motives of suicide, apart from the rationalizations concerned, are referred to and an important point is raised in this respect. An apparent marked improvement, amounting in some cases to an apparently dramatic recovery, may precede the suicidal attempt. The decision to commit suicide amounts to a partial solution of the unconscious conflicts involved in the genesis of the psychosis, hence the apparent improvement which causes the relatives to disregard medical advice.

H. W. EDDISON.

A Case of Symptomatic Epilepsy with Hysteroid Traits on a Background of Chronic Sepsis. (*Sov. Psychonev., No.* 3, 1936.) *Skvortzow, K. A., and Skipetrov, A. I.*

A case of a woman is described in which there were symptoms of subcortical disorder; peculiar oscillations of the body temperature, paroxysmal rises of blood-pressure, a sort of stupor induced by hyperventilation, delirious states, hypnagogic hallucinations, involuntary movements of the torsion type, as well as attacks of giddiness accompanied by peculiar visceral sensations. The discussion of this case touches upon many interesting problems in the pathological and pathogenetic interrelation of the symptoms enumerated. The authors consider the case to be an infective one with subcortical lesions of the brain.

(Authors' Abstr.)

Hypnosis and the Vegetative Nervous System. (*Sov. Psychonevr., No.* 3, 1936.) *Bogolepov, N. K.*

Susceptibility to hypnosis depends on many causes, and among them on the state of the vegetative nervous system. The author was able to confirm this by studying the degree of readiness to fall into hypnotic sleep of two groups of patients—alcoholics and hard smokers of tobacco. Whereas the former are vagotonics for the most part, the latter show symptoms of amphotonia with prevalence of sympathetic functions. It could be shown that the readiness of the patient to undergo hypnosis depends on his vegetative constitution, vagotonic patients being generally more easily hypnotized than the sympathicotonic ones. The patients most pliable to hypnosis are characterized by miosis, enophthalmos, pallor of the face, moist eyes, bradycardia, low blood-pressure, warm, moist hands, etc.

(Author's Abstr.)

Dispensary Observations on Morphinism. (*Sov. Psychonevr., No.* 3, 1936.) *Kantorovitsch, N. V.*

This is a report of the six years' experiences of the author with the treatment by dispensary methods of morphinomaniacs (125 cases carefully studied). The following conclusions of the author may be quoted:

(1) 48% of narcomaniacs had been absolutely healthy as to their neuro-mental condition before their disease began; the rest of the patients revealed different psychotic traits (psychasthenic, hysterical or unstable). No distinguishing characteristics in the bodily frame of narcomaniacs could be noted.

(2) The chief cause of the increase in the number of morphinomania cases was the imprudent and incorrect indication of treatment by physicians.

(3) Every man is liable to become a morphinomaniac under the influence of a prolonged intoxication with morphine. Psychopathic organization favours the origin of the condition, but it is not essential.

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(4) The existing methods of treatment give poor results (only in 11·4% of cases.) The great number of incurable cases makes it necessary to indicate for chronic patients the regular use of small doses of the drug. The author elaborates the indications and contra-indications, as also the dosage and the control for such a provision of chronic morphinomaniacs, which method of treatment gave favourable results as to the improvement in general condition and the working ability of patients. (Author's Abstr.)

Epidemic Encephalitis and Teaching Problems. (*Sov. Psychonevr., No.* 4, 1936.) *Bortnik, N. V.*

The author followed up 25 students of different higher schools suffering from sequela of epidemic encephalitis. In the progress made in their study, 11 of these students were compelled to abandon it; the other 15, with only slight post-encephalitis symptoms, chiefly vegetative disorders, continued their study, but showed slow progress compared with sane students. The author is of the opinion that even rudimentary post-encephalitic symptoms ought to serve as a contra-indication for the admittance of their bearers to study in higher schools.

(Author's Abstr.)

Neuropathic Patients in the Sanatoria for Nervous Cases. (*Sov. Psychonevr., No.* 4, 1936.) *Emdina, S., Voronova, M., and Asarkh, M.*

Among 2,477 patients cared for in two sanatoria there were 1,813 with different neuroses, and among the latter, 441 neuropathic subjects. The present paper deals with the authors' experiences during six months' work in the above-named sanatoria.

The following conclusions are reached:

(1) After a month's treatment at the sanatorium the neuropathic patients were considerably improved subjectively, especially those with hysteroid and schizoid reactions, and in a lesser degree neuropaths with psychasthenoid and epileptoid reactions.

(2) Somatic-vegetative changes in these subjects are of a permanent nature, and only slight improvement was seen in 40% of the patients.

(3) Amphotonic vegetative reactions in the hysteroid group of neuropaths were followed by a change into vagotonic reactions, whereas in the schizoids the change took place rather toward sympathicotonia.

(4–6) The blood-picture of neuropaths was characterized by a high degree of lymphocytosis. There was an increased content of calcium oxalate in the urine. Capillaroscopy revealed considerable and important alterations in the blood-vessels, which were of different nature in each group of neuropathic patients.

(7) Neuropaths are especially sensitive to atmospheric changes.

(Authors' Abstr.)

On the Nature of Psychasthenia. (*Sov. Psychonevr., No.* 4, 1936.) *Ozeretzkovski, D. S.*

On the base of a study of many cases of psychasthenia, several of which have been observed over a series of years, the author arrives at the following conclusions:

(1) The so-called psychasthenia cases, if one regards them from the standpoint of the new doctrine of "mild" forms of schizophrenia, may be ranged among these latter forms.

(2) Compulsive states may be considered as schizophrenic disorders of thinking; the so-called psychasthenic peculiarities of the personality are well explained from the same point of view as disturbances of activity and spontaneity in schizophrenia.

(3) Do all forms of the psychasthenia group belong to the mild forms of schizophrenia? This question cannot be solved at present, and its solution can be attained only with the assistance of the joint work of clinicians and psychologists.

(Author's Abstr.)

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On the Disorders of Orientation in Spatial Direction. (*Sov. Psychonevr., No.* 4, 1936.) *Vikker, J. L.*

Description of several cases in which the prominent symptom in patients was disorientation of spatial direction. The disorder that appeared at different times in a sort of single attack consisted in that all the surrounding objects seemed to the patient to be turned from left to right and vice versa by 180°. This is, of course, not a spatial disorientation in a psychiatric sense, nor is it an agnostic phenomenon or a disturbance of perception. The phenomenon must be considered as a manifestation of disorder in the representation of spatial direction. The consciousness of patients during the attacks was sufficiently clear. The duration varied from several minutes to one month (in one case). In one patient it was possible to provoke experimentally the shifting of objects by 45–90°. Here it was the appearance of a certain degree of drowsiness in the patient, who was made to look through a peep-hole in the screen of an apparatus adapted to provoke optokinetic nystagmus, that was largely responsible for bringing on the phenomenon of " turning ". This fact is of importance for the physiopathological interpretation of the latter. Ætiologically, we must consider alcoholism in the heredity of the patients, as also, in one patient, head injury, with a probable lesion of the frontal lobes. (Author's Abstr.)

The Selective Regional Vulnerability of the Brain and its Relation to Psychiatric Problems. (*Proc. Roy. Soc. Med., vol. xxix, p.* 1175, *July,* 1936.) *Meyer, Alfred.*

After summarizing the present position the writer points out that the globus pallidus has long been regarded as specifically affected in carbon monoxide poisoning. A similar condition occurs in poisoning with potassium cyanide and with ether. Lesions of the globus have been reported in respiratory failure, after morphine intoxication, after dinitrobenzol poisoning, and in a case of severe secondary anæmia. In all these conditions there is an interference with the oxygen transport to the tissues, causing inhibition of the respiratory enzyme system. The iron reaction of Spatz and the presence of calcium deposits are most marked in the globus.

The writer has found that ox brain minced cortex maintains its respiration on the addition of glucose, whereas this maintenance is almost entirely lacking in the globus, the cornu ammonis, the cerebellar cortex, and to a lesser degree in the striate body and the hypothalamus.

More vitamin C has been demonstrated in the cornu ammonis than in any other part of the brain. G. W. T. H. Fleming.

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4. Neurology.

Cerebral Lesions in Uncomplicated Fatal Diabetic Acidosis. (*Amer. Journ. Med. Sci., vol. cxcii, p.* 360, *Sept.,* 1936.) *Dillon, E. S., Riggs, H. E., and Dyer, W. W.*

The writers took as their criterion for coma a plasma carbon dioxide combining power of 20 vols. % or less. The brain in fatal diabetic acidosis shows lesions like those seen in acute asphyxia. The primary pathological changes occur in the cerebral capillary bed. The capillaries are dilated and the endothelial cells show degenerative changes with increased permeability of the walls, as evidenced by the presence of perivascular and pericellular œdema. There is proliferation of neuroglia, especially in the sub-ependymal and marginal areas, and acute degenerative changes in the ganglion-cells. The degeneration of the ganglion-cells is greatest in the third and fourth cortical layers, in the extrapyramidal system, in the cerebral vegetative centres of the diencephalon and of the medulla, in the olivocerebellar system, and in Sommer's sector of Ammon's horn. The choroid plexus is severely damaged. G. W. T. H. Fleming.

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Hereditary Progressive Bulbar Paralysis [*Contributo alla Forma Ereditaria della Paralisi Bulbare Progressiva*]. (*Riv. Sper. di Freniat., vol. lx, p. 1, March,* 1936.) *Vialetto, E.*

Three cases of progressive bulbar paralysis are described occurring in three sisters. Deafness was a marked feature in all three, associated in two with auditory hallucinations and persecutory delusions. The crossing of two recessives is shown in the family tree. H. W. EDDISON.

Subdural Traction and Post-traumatic Headache. (*Arch. of Neur. and Psychiat., vol. xxxvi, p. 75, July,* 1936.) *Penfield, W., and Norcross, N. C.*

Slight adhesions between the arachnoid and the dura are regarded as the cause of the persistent headache which follows cranial trauma. These adhesions tend to stimulate the sensitive dura. Insufflation of air is advocated and the technique is described. H. W. EDDISON.

Graves's Disease Complicated by Cranial Nerve Palsy. (*Sov. Psychonevr., No. 3,* 1936.) *Bogorad, F. A., and Retchnik, M. A.*

Description of three cases. In the first two there was a palsy of the oculo-motor nerves, due to changes in the oculomotor nuclei provoked by thyrotoxicosis. In the third case, that of a 42-year-old woman, the clinical picture was more complicated. There was an acute onset of the disease with headaches, nausea and ptosis, suggesting a cerebral affection. Later exophthalmos and tachycardia appeared. The thyroid gland remained of normal size. There was an increased cerebro-spinal fluid pressure. The authors assume in this case that the primary cause of the symptoms was an infectious disease, with possible affection of the sympathetic nervous system. (Authors' Abstr.).

On Multiple Cysticercosis of the Brain. (*Sov. Psychonevr., No.* 3, 1936.) *Stern, I. B., and Baskins, N. A.*

The authors discuss the symptomatology of the above-mentioned disease, and give a detailed description, with a histopathological analysis of a corresponding case of their own observation. This case is of interest because the diagnosis of cysticercosis could not be made here even retrospectively. Only the sudden death of the patient, as well as a certain degree of vagus aura of epileptic attacks in the form of vomiting, might have allowed one to suspect brain cysticercosis. The chief symptoms of this affection enumerated by the authors are: Prevalence of general cranial symptoms over the mild and hardly noticeable focal ones, intermittent course of the morbid phenomena with alternating periods of good health and severe headaches, vomiting and vertigo; acute frequently recurring psychotic episodes with many sudden false convalescences; accompanied by various epileptic fits and the paralytic curve, together with a strong pleocytosis and a negative Wassermann test of the cerebro-spinal fluid. (Authors' Abstr.).

Postural Hypotension. (*Arch. Int. Med., vol. lviii, p.* 773, *Nov.,* 1936.) *Ellis, L. B., and Haynes, F. W.*

The writers describe six cases of postural hypotension, and in five of the six there was involvement of the central nervous system. Postural hypotension results from a failure of the normal sympathetic vasomotor reflex to produce vasoconstriction when the subject assumes an erect posture. The centre controlling this reflex lies in the brain and not in the spinal cord. The lesion is either in a sympathetic centre or in an afferent pathway controlling the entire response, or it is generalized throughout the efferent pathways or nerve-endings. Ten out of seventeen successive patients with tabes dorsalis had an abnormal postural response of the blood-pressure.

Ephedrine was the most effective drug in relieving the symptoms; it probably acts by a central action on brain-cells or cerebral circulation. Ergotamine tartrate

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administered parenterally lessens the postural hypotension and may relieve the dizziness to some extent, but is not so effective as ephedrine.

G. W. T. H. FLEMING.

The Ætiology of Polyneuritis in the Alcohol Addict. (*Journ. Amer. Med. Assoc., vol. cvii, p. 642, Aug. 29, 1936.*) *Jolliffe, N., and Colbert, C. N.*

Twenty-eight alcohol addicts having uncomplicated polyneuritis were divided into three groups of seven, eight and thirteen patients. These three groups were given a basal diet containing quantities of vitamin B_1 estimated as slightly inadequate, twice, and four times the vitamin B_1 requirements respectively. These requirements were estimated by the use of Cowgill's vitamin/calorie ratio. Those receiving borderline amounts of vitamin B_1 showed no improvement in the objective symptoms of peripheral neuritis over a period of one month.

Two of these subjects were then given crystalline vitamin B_1 by intravenous injection, with dramatic response in one and a good response in the other. Subjects treated with a vitamin B intake approximately twice their predicted requirement showed improvement, but not as rapidly or in equal degree as those receiving four times their vitamin B requirement. All the subjects were given physical therapy, and symptomatic treatment was given as occasion required.

T. E. BURROWS.

Experimental Study of Alcoholism and Vitamin B Deficiency in Monkeys. (*Bull. Neur. Inst. New York, vol. v, p. 453, Aug., 1936.*) *Wechsler, I. S., Jervis, G. A., and Potts, H. D.*

Acute and chronic alcohol intoxication was induced in a group of monkeys as well as in cats fed on a normal diet. The clinical and pathological findings indicate that the nervous system of these animals offers great resistance to alcohol intoxication. A second group of four monkeys was fed on a diet either deficient in vitamin B complex or depleted of vitamin B_1. The clinical and pathological findings were comparatively mild, and in the main similar to those reported in the literature.

A third group of twelve monkeys was intoxicated with alcohol, and at the same time fed on a diet deficient in vitamin B complex or depleted of vitamin B_1. The clinical data and pathological findings point to a special pathogenicity of the combination as compared with the first and second groups. As far as peripheral nerves are concerned, no features recalling polyneuritis could be observed. In the third group, however, frequent vacuolization of the motor-cells of the anterior horns suggests incipient degeneration of the peripheral motor neurons.

T. E. BURROWS.

The Significance of Incomplete Homonymous Hemianopia in Brain Tumour. (*Bull. Neur. Inst. New York, vol. v, p. 201, Aug., 1936.*) *Johnson, T. H.*

The author presents a study of 49 cases of intracranial lesions causing incomplete homonymous hemianopia. All were verified at autopsy. The lesions include tumour, porencephaly, degeneration and trauma.

Among the frontal lobe cases 4 showed complete and 1 an incomplete homonymous hemianopia. Of the temporal lobe cases 7 had complete and 21 incomplete homonymous hemianopia. Of the 21 cases with an incomplete hemianopia 4 had quadrisectoral and 6 crescentic defects. In the occipital group 7 cases had complete and 4 incomplete homonymous hemianopia. Only one of the incomplete hemianopias was quadrisectoral in character.

It is concluded that homonymous sectoral field defects indicate a lesion in the visual system posterior to the chiasma, but do not locate the lesion in any specific area along this system. There are no homonymous hemianopic field changes which specifically represent involvement of any area of the brain. The proportion of incomplete to complete hemianopia is much greater in temporal lobe tumour than in occipital lesions. Frontal tumour rarely causes incomplete homonymous hemianopia. Crescentic defects are not as significant of occipital as of temporal

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lesions, and in the latter crescentic and quadrisectoral defects occur with equal frequency. T. E. BURROWS.

Visual Studies in Pituitary Adenoma. (*Bull. Neur. Inst. New York, vol. v, p.* 180, *Aug.*, 1936.) *Franklin, C. R.*

The visual findings in 28 verified cases of pituitary adenoma are analysed in an attempt to discover the factors of importance in diagnosis, and more so in prognosis in regard to post-operative effects on vision.

Fundi examination before operation showed 22 cases with changes in the papillæ ranging from slight temporal pallor to advanced primary optic atrophy. One case showed papillœdema. Failing vision was an initial symptom in 10 cases, and it was a symptom in all cases in which a reliable history was obtained.

The appearance of the papillæ seemed to be of little prognostic importance. The visual field defects were: bitemporal hemianopia, 21 cases; homonymous hemianopia, 2 cases; one eye blind and the other showing temporal hemianopia, 3 cases; and generally contracted fields, 2 cases. Of 10 cases with visual symptoms of less than two years' duration, 9 showed improvement of vision and 1 no change, whereas of 8 cases with symptoms of two to seven years' or longer duration, 3 improved, while 5 remained stationary or became worse. The degree of improvement taking place after operation is proportional to the visual acuity before operation.

The author observed that the visual fields disappeared in a clockwise manner on the right and in an anti-clockwise manner on the left, and that return of the fields occurred in a reverse order. The appearance of any marked degree of contraction in the fields seemed to be a bad prognostic sign. Improvement in the fields after operation was in general parallel to improvement in acuity, and was most marked in patients who were operated upon soon after the onset of visual symptoms.

X-ray therapy before operation did not appear effective in checking progressive failure of vision. The prognosis as regards post-operative restoration of fields and vision in pituitary adenoma is directly proportional to the early diagnosis and prompt surgical interference. T. E. BURROWS.

The Hearing in Patients with Intracranial Tumours. (*Bull. Neur. Inst. New York, vol. v, p.* 289, *Aug.*, 1936.) *Northington, P.*

The authors report 52 cases of intracranial tumours verified at operation or autopsy. The location of the tumours was 20 supratentorial and 32 subtentorial. 18 of the latter were acoustic neuromas; 3 others were situated in the cerebello-pontine angle.

It is not conclusive that the auditory function is affected except in tumours of the cerebello-pontine angle, but the probability of a small impairment of hearing for the upper two tones of the audiometer is suggested.

Clinical observation and studies on experimental animals as reported in the literature do not support the common assumption that the crossed auditory tracts are more important than the direct ones for auditory acuity. T. E. BURROWS.

The Differential Diagnosis of Parasagittal Gliomas and Parasagittal Meningiomas. (*Bull. Neur. Inst. New York, vol. v, p.* 218, *Aug.*, 1936.) *Masson, C. B.*

The author reports a series of 8 parasagittal gliomas and 19 meningiomas. Each hemisphere was affected with equal frequency. In a suspected parasagittal neoplasm where there is no X-ray evidence of a cranial hyperostosis, erosion or localized vascularity of the bone, and where air injection plates point to a parasagittal lesion if the initial symptom of the illness is a convulsive seizure, it is more likely to be a gliogenous growth than a meningioma. The presence of papillœdema favours the diagnosis of glioma, and the lesion is more likely to be a relatively benign type in the frontal lobe.

All the parasagittal gliomas began with a convulsive seizure, whereas only 50% of the meningiomas did so. T. E. BURROWS.

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Angioblastic Meningiomas. (*Bull. Neur. Inst. New York, vol. v, p.* 485, *Aug.*, 1936.) *Wolf, A., and Cowen, D., jun.*

The authors present 6 cases of supratentorial angioblastic tumours. While these tumours all possess the gross appearance of an ordinary meningioma, they vary histologically from types indistinguishable from the typical cerebellar hæmangioblastoma to those with distinctly meningiomatous features. Histological evidence, therefore, as well as their gross appearance justifies the term "angioblastic meningioma". T. E. BURROWS.

Migraine: A Critique of the Hypophyseal Theory. (*Bull. Neur. Inst. New York, vol. v, p.* 437, *Aug.*, 1936.) *Timme, W.*

The migrainous attack occurs in a certain special type of constitution, namely the so-called status hypoplasticus of Bartels, consisting primarily of a hypoplasia of the hypophysis, cardiovascular system, gonads and adrenals. The symptoms of migraine are the result of the response of the hypoplastic constitution to critical periods.

The large variety of symptoms may be grouped into three classes: First, those produced by pressure of an expanding pituitary within too small a sella turcica; secondly, those produced by the effect of the hypophyseal activity on the various organs and tissues; and lastly, those produced by the lack of pituitary control in hypoplasias, as the gland endeavours vainly to reach an activity produced by too great a demand upon it, thereby bringing from a latent state to a visible one such symptoms as allergic œdemas, anginas, purpuras and hæmorrhages.

The rationale of the disappearance of migraine in later life is the enlargement of the sella through frequent recurring pressure together with gradual shrinking of the gland itself. There is a migrainous equivalent which then arises—a periodic disturbance of sleep, with accompanying symptoms much resembling the earlier migrainous ones, affecting water exchange, blood-pressure, mental state, gastro-intestinal system and the sympathetic nervous system. T. E. BURROWS.

The Development and Constituents of the Human Hypophysis. (*Bull. Neur. Inst. New York, vol. v, p.* 387, *Aug.*, 1936.) *Tilney, F.*

The author utilized fourteen stages of human development from the 11-mm. human embryo to the adult.

The human hypophysis, following the general rule in mammals, has a two-fold ectodermic origin. The oral ectoderm, through the formation of Rathke's mouth-pouch, gives rise to the glandular lobe; the neural ectoderm forming the infundibular region of the third ventricle gives rise to the neural lobe.

Histological and topographical specializations in the glandular lobe determine three distinct subdivisions, i. e., (1) the pars tuberalis, (2) the pars infundibularis, and (3) the pars distalis. Each one of these three glandular subdivisions follows a specific mode of development. The pars tuberalis has its inception in two lateral diverticula, which make an early appearance as two lateral buds, one on either side of the median portion of the original mouth-pouch of Rathke.

The tips of these lateral diverticula turn inward beneath the median eminence of the tuber cinereum, and ultimately fuse across the median line to form the pars tuberalis. Two caudal prolongations from the original mouth-pouch extend backward to surround the infundibular region. These caudal prolongations also fuse across the midline to form the pars infundibularis. The pars distalis takes origin in the median portion of the mouth-pouch. Unlike the pars tuberalis and pars infundibularis, it is not in contact with any portion of the neural lobe. It forms the bulk of the pituitary gland and occupies a position deep in the sella turcica. The pars distalis consists of a medullary core and a cortical zone which almost completely surrounds this core. The basis for this distinction is laid down in early embryonic life. While it is definite that the tips of the lateral processes give rise to the pars tuberalis, the proximal portions of these lateral processes form

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a broad zone about the median portion of the gland. Early in the course of development a deep groove running along either side of the dorsum of the original mouth-pouch establishes a boundary between the median and lateral portions of the pars distalis. The subsequent ingrowth of blood-vessels and connective-tissue trabeculæ through these grooves emphasizes this subdivision into a medullary cone and cortical zone. Topographical specialization and specific relations to glandular portions of the pituitary determine four distinct subdivisions of the neural lobe, i. e., (1) the median eminence of the tuber cinereum, (2) the infundibulum, (3) the infundibular stem, and (4) the infundibular process. The terms for the different parts of the hypophysis suggested are based on strictly morphological implications.

T. E. BURROWS.

Occlusion of the Aqueduct of Sylvius by Neoplastic and Non-neoplastic Processes, with a Rational Surgical Treatment for Relief of the Resultant Obstructive Hydrocephalus. (*Bull. Neur. Inst. New York, vol. v, p.* 348, *Aug.*, 1936.) *Stookey, B., and Scarff, J.*

Progressive occlusion of the aqueduct of Sylvius may be caused by either non-neoplastic or neoplastic processes in the tissues surrounding the duct.

The appearance of these processes in persons other than infants born with congenital anomalies of the aqueduct is rare. The authors have been able to collect from the literature only 22 cases, of which 16 were non-neoplastic. To this series they have added 6 cases of their own, of which 4 were non-neoplastic.

The essential clinical feature of these cases has been the evidence of an obstructive hydrocephalus associated with clinical signs, suggesting in part a suprasellar cyst, and in part a midline tumour of the cerebellum.

In the majority of cases diagnosis can only be confirmed by air studies and dye tests. The authors' treatment consists of draining the obstructive fluid out of the ventricles directly into the large subarachnoid cisternæ at the base of the brain by means of openings through the lamina terminalis and the floor of the third ventricle.

The authors have performed this operation on 6 cases. Four of these cases were improved, and 3 of them, belonging to the non-neoplastic group, have remained symptom-free for 3 years, 14 months and 6 months respectively after the operation. These cases would appear to be permanently cured. T. E. BURROWS.

The Syndrome of the Unruptured Aneurysm of the Intracranial Portion of the Internal Carotid Artery. (*Bull. Neur. Inst. New York, vol. v, p.* 247, *Aug.*, 1936.) *McKinney, J. McD., Acree, T., and Soltz, S. E.*

The authors report 8 cases, and review 29 cases collected from the literature. They conclude that the classical syndrome of an unruptured aneurysm of the intracranial part of the internal carotid artery consists of exophthalmos, and a partial or complete involvement of the second, third, fourth, fifth and sixth cranial nerves. The third and fifth are always involved. This syndrome is not pathognomonic of aneurysms in this region, nor do all these aneurysms give rise to this syndrome.

X-ray evidence of bony destruction of the sella turcica on the side corresponding to the findings, with or without linear streaks of calcification, is conclusive evidence of this condition.

Cases in which there is difficulty in the differential diagnosis can usually be clarified by careful encephalography. T. E. BURROWS.

Visual Disturbances of Obscure Ætiology Produced by Focal Intracranial Lesions Implicating the Optic Nerve. (*Bull. Neur. Inst. New York, vol. v, p.* 223, *Aug.*, 1936.) *McKendree, C. A., and Doshay, L. J.*

Six cases having loss of vision as the outstanding symptom and without the usual accompanying signs or symptoms that make possible a precise clinical interpretation are described.

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The lesions found in three cases were aneurysms arising from the internal carotid artery or one of its branches.

In the remaining three cases, meningiomas arising from the edge of the optic foramen, olfactory groove and the sphenoid ridge were discovered. It is important to consider the possibility of the existence of intracranial expanding lesions in all obscure cases of visual defect, with or without optic atrophy, regardless of obvious focal infection, systemic symptomatology, or psychoneurotic disturbances. Early recognition of the lesion may lead to the preservation of vision and life.

T. E. BURROWS.

The Effects of Lesions of the Superior Cerebellar Peduncle in the Macacus rhesus Monkey. (*Bull. Neur. Inst. New York, vol. v, p.* 165, *Aug.*, 1936.) *Ferraro, A., and Barrera, S. E.*

The authors describe some of the symptoms associated with experimental lesions of the superior cerebellar peduncles in 23 *Macacus rhesus* monkeys. In most of the cases involving only the superior cerebellar peduncle the lesions were incomplete, and if bilateral, of different extent on the two sides. Such incomplete unilateral cases showed ipsilateral dysmetria with more or less hypotonia and hypo-reflexia, the last two elements being more marked in the hind legs.

In bilateral cases the same symptoms were found on both sides.

These symptoms were transient, almost completely disappearing by the end of a few weeks.

In one case a complete unilateral punching-out of the superior cerebellar peduncle, without any complicating features, resulted in ipsilateral dysmetria and hypotonia, together with rolling movements towards the side of the lesion, nystagmus to the opposite side, postural asymmetries and spiralling towards the side of the lesion on movements such as climbing a pole and swimming. In the postural asymmetries the chin was turned toward the side of the lesion, and there was a relative ipsilateral flexion and contralateral extension of the limbs. There was also a marked spontaneous resting tremor in the head and ipsilateral limbs. The authors suggest that there are, therefore, fibres of vestibular origin at various levels in the superior cerebellar peduncles.

Bilateral lesions of the cerebellar peduncles alone, although of different extent on the two sides, showed symmetrical involvement in the symptoms.

A small lesion in the peduncle is capable of producing cerebellar symptoms, but it apparently requires a complete lesion to produce the vestibular component in the symptoms.

Spontaneous resting tremor was very severe in one case of complete destruction of one superior cerebellar peduncle, but the same variety of tremor was seen whenever some complicating lesion involved other structures in addition to a partial lesion of the superior cerebellar peduncle. T. E. BURROWS.

Observations on the Nervous System of Bdelloura Candida. (*Bull. Neur. Inst. New York, vol. v, p.* 141, *Aug.*, 1936.) *Elwyn, A.*

The nervous system of *Bdelloura candida* has the form of a flattened orthogon composed of three pairs of longitudinal trunks connected by rings of transversely running nerves. The ventral half is strongly developed, the dorsal greatly reduced in size.

The brain consists of a dorsal unsegmented portion related to nerves N1–N5 and to the optic nerve, and a ventral part representing the fusion of at least three ganglionic segments of the ventral nerve-cord. The ventral commissures and ventrolateral nerves contain ganglion-cells, and, together with the brain and main nerve-cords, must be regarded as continuous parts of a widely distributed central nervous system. Histologically there is a differentiation into a peripheral ganglionic layer and a central fibre-mass, resembling the conditions found in Annelids.

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This arrangement is most obvious in the brain, but is also indicated in the ventral nerve-cord, and to a lesser extent in the transverse ventral nerves.

A glial supporting tissue is well organized, furnishing a definite fibrillar covering for the brain and main nerve-trunks, and extending into the interior in the form of a spongy network. T. E. BURROWS.

Rhythmic Myoclonus of the Muscles of the Palate, Larynx and Other Regions: A Histo-pathologic Study. (*Bull. Neur. Inst. New York, vol. v, p. 94, Aug.*, 1936.) *Davison, C., Riley, H. A., and Brock, S.*

The authors record a case of rhythmic myoclonic movements of the ocular, oral, palatal, pharyngeal and laryngeal muscles. There was destruction of the right central tegmental tract, medullary substance of the right cerebellar hemisphere, slight involvement of the right dentate nucleus and pseudo-hypertrophy of the right inferior olivary complex.

From a study of this case and others previously recorded, it is concluded that the lesion which permits the appearance of these movements is usually confined to the ipsilateral central tegmental tract and the ipsilateral inferior olivary nucleus.

In a few cases the lesions were situated in the dentate nucleus and contralateral inferior olivary nuclei.

Most of the time the movements were on the side contralateral to the lesion of the central tegmental tract or of the inferior olivary nucleus. In some of the few cases with lesions of the dentate nucleus, the movements were on the ipsilateral side. T. E. BURROWS.

A Pathognomonic Encephalographic Sign of Subdural Hæmatoma. (*Bull. Neur. Inst. New York, vol. v, p. 135, Aug.*, 1936.) *Dyke, C. G.*

The author presents a case of subdural hæmatoma in which a unique encephalographic picture was obtained.

This consisted of a large finger-like collection of air over the cerebral hemisphere projecting diagonally, ventrally and laterally from the mid-line.

The air was considered to lie between the arachnoid and the inner membrane of the hæmatoma.

It is pointed out that this picture was unique in a series of 3,500 air studies, of which only 6 were cases of subdural hæmatoma.

It is suggested that if encephalography were more frequently used in potential subdural hæmatoma cases, the picture described might be encountered more often. T. E. BURROWS.

Some Neurological Syndromes Produced by Arsenic and Lead. (*Bull. Neur. Inst. New York, vol. v, p. 28, Aug.*, 1936.) *Cornwall, L. H.*

The author reports six cases of poisoning by arsenic and lead.

Prominent symptoms apart from those due to polyneuritis were headache, worse at night—in one case this was the only symptom—fatigue, mental confusion, and in one case a labile blood-pressure which varied from 138/82 to 170/95.

In every case both lead and arsenic were found in the urine.

The quantity of arsenic found in the different cases varied from 0·071 to 2·84 mgrm. per 100 grm. of dried specimen, and the quantity of lead from 0·03 to 0·37 mgrm. per 1,000 c.c.

The urinary content indicated merely the amount of metal that was being eliminated at the time that the specimen was collected, and had no relation to the clinical condition of the patient.

It is pointed out that an absence of metal in the urine may mean nothing unless the patient is receiving eliminative therapy at the time that the specimen is collected.

The treatment in each case consisted in the administration of sodium thiosulphate orally and, in some cases, by the intravenous route as well.

All the cases recovered. T. E. BURROWS.

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Sensory and Other Aspects of Multiple Sclerosis. (*Bull. Neur. Inst. New York, vol. v, p. 16, Aug.*, 1936.) *Brickner, R. M.*

Sixty-two cases of multiple sclerosis were systematically, and 40 cases less formally, observed over a varying period of years. Both subjective and objective sensory changes were present in more than 95% of the patients.

The commonest objective defect was due to lesions of the posterior columns, and occurred in 95% of the cases.

Implication of the spino-thalamic tract occurred in 38·7%, and "numbness" was a first symptom in 24·2% of cases. Pain occurred in almost 100% of patients, but was frequently of mechanical origin.

Euphoria occurred in only 10 cases and 9 cases were depressed.

The most frequent first symptoms were gait disturbance 22 cases, numbness 15 cases, and diplopia 13 cases.

Other symptoms described are facial paralysis, sphincter involvement and neurosis. T. E. BURROWS.

Neoplastic Cysts Communicating with the Lateral Ventricles. (*Bull. Neur. Inst. New York, vol. v, p. 21, Aug.*, 1936.) *Cohen, I.*

A case of hæmangiomatous cyst and one of astrocytomatous cyst communicating with the lateral ventricle are reported. Evidence of bleeding into the cysts as shown by changes in the cerebro-spinal fluid withdrawn by lumbar puncture was found in both cases.

Air injected through the lumbar route filled the cysts.

The ventricles were dilated, but not displaced. T. E. BURROWS.

Convolutional Digitations Seen in the Rœntgenograms of Immature Human Skulls. (*Bull. Neur. Inst. New York, vol. v, p. 61, Aug.*, 1936.) *Davidoff, L. M.*

The convolutional impressions of the brain upon the inner table of the skull as seen in rœntgenograms were studied in about 2,500 normal individuals varying from three months up to eighteen years of age.

It was found that these markings when charted in terms of experimental units for the various ages produced certain characteristic curves.

By comparison with the growth curves of the brain at corresponding ages there appeared to be a similarity in the two curves up to the age of eight years. The disparity beyond this point can be accounted for by changes in the thickness of the skull, associated with variations in the mineral reserve during the growth of the skeleton as a whole. T. E. BURROWS.

Clinical Diagnosis of the Tumours of the Corpus Callosum. (*Bull. Neur. Inst. New York, vol. v, p. 37, Aug.*, 1936.) *Cramer, F.*

The author presents six cases of glioma originating in the corpus callosum.

In two cases the tumour was situated in the genu, in two cases in the body and in two cases in the splenium. All were verified at autopsy.

Headache occurred as the first symptom of each of the two cases in which the tumour occupied the body and anterior part of the corpus callosum, and in them it was very severe. In those in which the tumour was located in the genu headache became severe only towards the end of the illness, and in the cases involving the splenium headache was not mentioned in the history.

Projectile vomiting occurred in all the cases except one with astrocytoma of the splenium.

In both cases of involvement of the splenium papillœdema was less marked than in the other cases.

Epileptiform seizures occurred in one case in which the tumour was in the genu and anterior portion of the body.

Cataplectic attacks occurred in two cases, one of tumour of the genu and one of tumour of the anterior part of the body.

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Vertigo was present only in the two cases involving the body.

Unsteadiness of gait was severe in tumour of the splenium, in which also loss of memory for recent events was prominent.

Head noises occurred in three cases, one of involvement of the genu and two of the body.

Incoherent speech was present in all the patients in the absence of motor or sensory aphasia *per se*.

Inattention and apathy were most marked in tumours of the genu, while tumours of the splenium produced pronounced mental confusion. The latter cases showed the maximum disturbance of equilibration, without disturbance of synergy of individual movements.

Apraxia involving especially the left hand occurred in all the cases.

All the patients were right-handed.

Some of the mental signs encountered were agnostic in nature.

T. E. BURROWS.

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5. Pharmacology and Treatment.

Involutional Melancholia. (*Arch. Neur. and Psychiat., vol. xxxv, p.* 1076, *May*, 1936.) *Werner, A. A., et al.*

The writers, in continuation of previous work, treated cases of involutional melancholia with theelin (1 c.c. of the aqueous solution daily for six months). They found that more than 50% of cases of uncomplicated involutional melancholia showed marked improvement; a further 20% showed moderate improvement. Theelin also shortens the period of mental illness. A history of previous mental illness in the patient suggests a guarded prognosis. G. W. T. H. FLEMING.

Attempts at the Treatment of Schizophrenia with Tissue Histolysates of Tushnov. (*Arch. Sci. Biol.* [*U.S.S.R.*], *vol. xxxvii, pp.* 193–211, 1935.) *Maiorov, F. P., Vinogradov, N. V., Golovina, V. P., Narbutovich, I. O., and Povorinskii, Y. A.*

Seventeen patients were treated. Positive results are claimed in five cases and none in the others. W. A. PERLZWEIG (Chem. Abstr.).

The Value of Sulphur Pyretotherapy in Schizophrenia [*Il valore della zolfopiroterapia nella schizophrenia*]. (*Riv. Sper. di Fren., vol. lx, p.* 205, *June*, 1936.) *Imber, I.*

It is concluded that no beneficial result is obtained from this treatment beyond a sedative effect upon restless subjects. The technique employed consisted in injecting powdered sulphur 2% in camphorated olive oil to which 0·4% novocaine was added. The optimum dose for each case was ascertained and injected intramuscularly into the glutei at intervals of a week, 10 to 16 injections being administered. H. W. EDDISON.

Early Operative Interference in the Treatment of Certain Psychoses. (*L'Encéphale, vol. ii, 31st year, p.* 1, *June*, 1936.) *Moniz, E.*

The early operative attempts on psychoses seemed unreasonable, and took the form merely of further disturbing an already disordered brain.

Attention here has been directed entirely to the pre-frontal area. Of particular importance has been the observation that greater psychic disturbance occurs with lesions of the white matter in the pre-frontal area than with lesions of the grey. This is in accord with the findings of Strauss and Keschner that meningiomata produce less mental disturbance than gliomata, and especially the spongioblastomata.

In order to obtain portions of the centrum of the pre-frontal lobes an instrument has been devised. This consists of a cannula closed at one end and having a slit

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close to the closed end. A steel stylet in the cannula is caused to bulge out of the slit when the stylet is pressed down. The cannula is introduced through a trephine hole, pushed into the brain and pressure is made on the end of the stylet. It then protrudes from the slit in the cannula and, on rotating the cannula, section is made of the nervous tissue. This device has been called the leucotome, and the operation leucotomy. An alternative has been to inject the area with absolute alcohol.

The cases selected were of all types, but specially agitated melancholics. There were no deaths in the series, and none of the patients were rendered worse by the experiment. A series of 20 cases is reported.

The conclusions were that destruction of certain pre-frontal areas produces remarkable alterations in psychic symptoms. The connection between cerebral lesions and mental symptoms appears to the author to be close.

W. Mc. Harrowes.

Dynamics of Schizophrenia under the Influence of Prolonged Narcosis. (*Sov. Psychonevr.*, 1936, *No.* 4.) *Gilarovski, V. A.*

The author studied the influence of prolonged narcosis in schizophrenic patients, with the object of determining the optimal conditions for the attainment of therapeutic effects, and the changes which take place in the somatic and mental sphere and in the metabolism. The present paper contains a detailed description of all these changes, with an attempt to explain the underlying mechanism of them.

During the period of falling asleep an increase of new hallucinations of schizophrenic type was observed, as also delirious states, the toxic nature of which is demonstrated by the appearance of similar states in mescaline intoxication and in hypoglycæmic shock. Of interest also are the emotional states observed after awakening. They are of euphoric nature (although the mood of the patient may sometimes be dull and uneasy). Sometimes the entire behaviour of patients changes; a tendency to joke, to speak in rhymes, to play the fool is observed, but this state is not hebephrenic, and is only due to a decrease of inhibition causing a greater motor activity.

As to the mechanism of the action of narcosis, the author is of the opinion that this action is subcortical. We may even speak of a sort of diencephalosis, since there are vegetative disorders, such as hyperæmia of the face, increased perspiration, salivation, respiratory disorders, increased temperature of the body, pupillary symptoms, besides the sleep itself. Epistaxis, urticaria and sometimes premature appearance of menses were also observed. (Author's Abstr.)

The Toxicity of Barbital Derivatives. (*Journ. Amer. Pharm. Assoc., vol. xxv, pp.* 597–601, 1936.) *Jones, I., and Lynn, E. V.*

The toxicity in rabbits and the comparative efficiency were studied for amytal, barbital, dial, neonal, pentobarbital, phanodorm and phenobarbital, with both oral and intraperitoneal routes. Some of the figures for the minimum lethal dose differ markedly from those found in the literature. Apparently the general ratio of efficiency to toxicity is approximately the same for the seven compounds, except that amytal and pentobarbital seem to be extremely safe by oral administration.

A. Papineau-Couture (Chem. Abstr.).

Presence of Strychnine-Barbituric Complex in the Urine of Animals which had Received Separate Injections of Strychnine and a Barbiturate. (*Compt. Rend. Soc. Biol., vol. cxxi, pp.* 1412–13, 1936.) *Lavergne, V. de, Kissel, P., Weiller and Chahidi, H.*

Strychnine was injected into one leg of a guinea-pig and a barbiturate (soneryl or gardenal) into another leg. The strychnine-barbituric complex previously described by de Lavergne and Kissel (cf. *Bull. Acad. Med.*, vol. cxiv, p. 384, 1935) was extracted from the urine collected in the next twenty-four hours. The toxicity of the complex is relatively low. L. E. Gilson (Chem. Abstr.).

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Chronic Experimental Barbiturism. (*Semana Med.* [*Buenos Aires*], *vol. ii, pp.* 304–9, 1936.) *Carratala, R. E.*

Barbital, somnifaine and dial were tested on dogs. A dose which produced within 30 minutes a light narcosis and six hours' sleep was applied daily for two months. The animals showed some nervous symptoms; one developed a tolerance; none died. In a second series the dose was doubled every 6 days. The dogs developed a high tolerance, but died finally after 44 to 57 days. Severe nervous symptoms developed. The autopsy revealed congestion in heart, intestines, kidneys and liver. A. E. MEYER (Chem. Abstr.).

Comparative Actions of Various Narcotics. IV. The Narcotic Effects of Cyclohexane and Benzene. (*Journ. Physiol.* [*U.S.S.R.*], *vol. xx, pp.* 142–5, 1936.) *Brusilovskaya, A. I., and Lazarev, N. V.*

Contrary to the generally accepted view, experiments show that cyclohexane is a more powerful narcotic than benzene.

Ibid. V. Effect of Lengthening or Branching of the Hydrocarbon Chain. (*Pp.* 146–55.)

Toluene is a more powerful narcotic than benzene, whereas xylene is still more powerful than toluene. As to aliphatic hydrocarbons, if the concentration of these substances in the blood is computed on the basis of a true aqueous solution, an increase in the size of the hydrocarbon molecule brings about an increase in the narcotic effect (in harmony with Richardson's rule). Just what effect the forking of hydrocarbon chains has on the narcotic action (i.e., pentane and isopentane) has not been definitely established.

Ibid. VI. Effect of Introducing a Halogen into a Hydrocarbon. (*Pp.* 156–60.)

The powerful narcotic effect shown by chlorinated hydrocarbons, when compared to unsubstituted hydrocarbons, is due almost entirely to the greater solubility of the former in water and in the blood.

The prevailing opinion is that methyl and ethyl alcohols are almost typical narcotics, whereas CH_4 and C_2H_6 are very weak narcotics. Actually just the opposite is the case, if the effective narcotic concentration of these substances in the blood is taken into consideration. The narcotic action of hydrocarbons is greatly diminished on the introduction of the first HO group.

H. COHEN (Chem. Abstr.).

Detoxification of Dendrobine by Sodium Amytal. (*Proc. Soc. Exptl. Biol. Med., vol. xxxiv, pp.* 553–4, 1936.) *Chen, K. K., and Rose, C. L.*

Dendrobine, an alkaloid of the Chinese drug chin-shih-hu, causes convulsions of central origin. In the rabbit the subcutaneous minimum lethal dose of dendrobine HCl is 100 mgrm. per kgrm. of body-weight and 17 mgrm. when injected intravenously. The repeated intravenous injection of sodium amytal in doses of 25 and 12·5 mgrm. per kgrm. antidoted 5 m.l.d. of the alkaloid after definite signs of intoxication had appeared. C. V. BAILEY (Chem. Abstr.).

The Effect of Hypnotics on Picrotoxin: A Contribution to the Problem of the Cold Centre. (*Arch. Exptl. Path. Pharmakol., vol. clxxxi, pp.* 219–32, 1936.) *Rosenthal, F., and Wallach, G.*

Morphine, chloralose, paraldehyde, chloretone, phenobarbital and barbital minimize or prevent the fall in body temperature caused by the subcutaneous injection of picrotoxin (1 mgrm./kgrm.) into rabbits. Chloral hydrate and urethane are ineffective, the latter despite the fact that it inhibits the hyperpyrexia caused by β-tetrahydronaphthylamine. There is no significant difference in the efficacy of cortical and brain-stem hypnotics. The hyperglycæmia caused by picrotoxin is also inhibited by these hypnotics. HARRY EAGLE (Chem. Abstr.).

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Relative Strengths of Various Narcotics. VIII. Substitution Series of Narcotics. (*Journ. Physiol.* [*U.S.S.R.*], *vol. xx, pp.* 906–26, 1936.) *Brusilovskaya, A. I., and Lazarev, N. V.*

When the H of a hydrocarbon is replaced by various substituents, the resulting substance will be more strongly narcotic the less polar the molecule becomes; the narcotic strength is, in addition, dependent on the size of the molecule. The narcotic strength of radicals is given in the following " substitution series ": Alkyl radical $\left\{ \begin{array}{l} \\ \end{array} \right.$ >polymethylene radical >aryl radical >alkyl radical, which contains multiple bonds (the more such bonds, the weaker) > $-H > -I > -Br > -Cl > -NH_2$, $-SH$, $-CHO$, $-OH$, $-NO > -COOH$. Thus, the replacement of a hydroxyl group of Cl brings about an increase in the narcotic strength of the substance. The exact location of the radicals NH_2, $-SH$, $-CHO$, $-OH$, $-NO_2$ in the series is still undetermined.

Ibid. X. The State of the Narcotic in the Blood. (*Pp.* 939–44.) *Lazarev, N. V.*

Some narcotics are more soluble in water than in the blood (e.g., ethyl ether, acetone, N_2); others are more soluble in blood than in water (ethylene, ethyl chloride, chloroform, benzene). A part of the narcotic is therefore present, not in the form of a true aqueous solution, but is " bound " (i.e., adsorbed by the plasma proteins or erythrocytes, dissolved in fats or lipoids, etc.). The values of the ratio " bound " narcotic to water-soluble narcotic in the blood are N_2 0·13, acetylene 0·2–0·3, N_2O 0·2, Et_2O 0·2, methane 0·5, ethylene 0·8–1·25, EtCl 0·8–2, CCl_4 0·8–2, $CHCl_3$ 3·3, benzene 0·8, cyclohexane 4·4, pentane 11, hexane 22, heptane 53·5. It may be assumed that weak narcotics are present in the blood in the form of a true aqueous solution, whereas strong narcotics are completely " bound ".

H. COHEN (Chem. Abstr.).

Pharmacological Study of Pernocton. (*Giorn. Ital. Anestesia Analgesie, vol. ii, pp.* 73–89, 1936.) *Mannini, R.*

In rats pernocton subcutaneously gave a marked hypnotic and few secondary effects. Intravenous or intramuscular injection into rabbits and dogs showed the hypnotic dose to be far less than the toxic dose. Toxicity was increased with speed of injection. Animals under pernocton hypnosis were highly tolerant to Et_2O and $CHCl_3$.

H. L. GRUEHL (Chem. Abstr.).

Narcosis and Glutathione. (*Giorn. Ital. Anestesia Analgesie, vol. i, pp.* 375–83, 1935.) *Galli, G., and Mingazzini, I.*

After short narcosis (10 minutes, 30 minutes) of rabbits with $CHCl_3$, Et_2O, C_2H_4 and N_2O there was a diminution of reduced glutathione in the lung-tissue, an increase in the liver tissue and a diminution from the blood. Kidney and brain determinations varied. Twenty-four hours after the narcosis there was an increase in reduced glutathione, particularly in the animals which had received N_2O.

H. L. GRUEHL (Chem. Abstr.).

Narcosis and the Dehydrogenation Processes. (*Giorn. Ital. Anestesia Analgesie, vol. i, pp.* 315–27, 1935.) *Galli, G., and Radici, G.*

Cellular metabolism (brain, lung, liver and kidney of rabbits) was little affected by short narcosis with chloroform, ether, C_2H_4 or N_2O. In general the gases caused less alteration than $CHCl_3$ or Et_2O. After twenty-four hours the R.Q. of the organs returned to normal.

H. L. GRUEHL (Chem. Abstr.).

Some Effects of Pentobarbital on the Rabbit. (*Proc. Soc. Exptl. Biol. Med., vol. xxxiv, pp.* 65–7, 1936.) *Blackberg, S. N., and Hrubetz, M. C.*

Pentobarbital in doses of 40 mgrm. per kgrm. of body-weight was injected intraperitoneally, and at the time of maximum depression 10 c.c. of 50% glucose solution was slowly injected intravenously. In the normal-fed animal the glucose did not

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appreciably shorten the period of depression. Fasting for twenty hours greatly increased the duration of the depression. There was no correlation between the blood-sugar levels and the susceptibility to the drug. At the time of maximum depression the blood-sugar level showed increased variability and there was a definite drop at the time of recovery, a drop greater than could be explained by inanition. C. V. BAILEY (Chem. Abstr.).

Comparative Minimal Hypnotic Effects, Toxicity and Pathology Produced by Sodium and Magnesium Salts of Phenobarbital. (*Proc. Soc. Exptl. Biol. Med., vol. xxxiii, pp.* 621–4, 1936.) *Taylor, W. F., and Lackey, R. W.*

There is no essential difference in the toxicity of the sodium and magnesium salts of phenobarbital when administered orally to the rat or the dog. In the dog magnesium phenobarbital administered intravenously in doses of 30 mgrm./kgrm. produced a more pronounced hypnotic effect than did the sodium compound. It is apparent that the additive depressing effect of the magnesium ion, as opposed to the stimulating effect of the sodium ion, is responsible for the difference in degree of hypnosis of the two salts. No such effect was observed when the two salts of phenobarbital were adminstered orally. C. V. BAILEY (Chem. Abstr.).

Effects of Adrenaline on the Cerebral Circulation. (*Compt. Rend. Soc. Biol., vol. cxxii, pp.* 365–9, 1936.) *Cachera, R., and Fauvert, R.*

A glass window was inserted in the skull of a dog and photomicrographs of the cortical vessels taken at 5-sec. intervals. The injection of 0·1–0·2 mgrm./kgrm. of adrenaline into the circulation caused a prompt increase in the diameter of the cerebral arteries and veins. L. E. GILSON (Chem. Abstr.).

Effects of Adrenaline on the Cerebral Circulation of the Yohimbinized Dog. (*Ibid., pp.* 381–5.)

After injection of yohimbine the effects of adrenaline on the diameter of the cerebral vessels were irregular. L. E. GILSON (Chem. Abstr.).

The Action of Thiocyanogen Compounds on the Central Nervous System. (*Folia Pharmacol. Japon., vol. xxii, pp.* 157–71, 1936.) *Moriki, H.*

Experiments with a series of organic thiocyanogen compounds as well as with sodium rhodanate in mice showed that these compounds intensified the reaction of central stimulants such as strychnine and caffeine. The morphine tail reaction was produced by much smaller doses in the presence of the rhodanate, and convulsions accompanied the administration of chloral hydrate and urethane. The calcium-ion content of the blood, the blood-pressure and temperature remained unchanged, but reflex excitability of the cord was greater.

G. H. W. LUCAS (Chem. Abstr.).

The Central Action of Acetylcholine. (*Journ. Pharmacol., vol. lvi, pp.* 448–50, 1936.) *Silver, Geo. A., and Morton, G.*

Acetylcholine in doses of 2 to 4γ injected into the hypothalamic region of cats anæsthetized with urethane caused a small rise in blood-pressure, followed by a sharp fall lasting from 1 to 2 minutes. Similar injection into the lateral ventricle gave the same effects less consistently. Intraventricular injections into the unanæsthetized cat caused drowsiness lasting about 1 hour.

T. H. RIDER (Chem. Abstr.).

Biochemistry of Choline and Its Derivatives. IV. Action of Blood on Several Quaternary Ammonium Bases with Ester Groups. (*Bull. Soc. Chim. Biol., vol. xviii, pp.* 529–55, 1936.) *Kahane, E., and Levy, J.*

Propionylcholine, formylcholine and acetylcholine are rapidly hydrolyzed by the esterase in the blood, and their muscarine-like action seems to be proportional

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to their rates of hydrolysis. Eserine sensitizes the dog to their action. Betaine ethyl ester, benzoylcholine and acetyl-methylcholine are hydrolyzed slowly in the blood, and stearylmethylcholine, isovalerylmethylcholine, bromocholine, carbamylcholine and choline HNO_3 ester not at all. All have a much weaker hypotensive action than acetylcholine, and eserine does not increase their action.

L. E. GILSON (Chem. Abstr.).

The Antagonism Between Curarine and Acetylcholine. (*Journ. Physiol., vol. lxxxvii, pp.* 425–8, 1936.) *Briscoe, G.*

In partial curarization the effect of large doses of acetylcholine is reversed and temporary augmentation of contraction is seen instead of depression.

E. D. WALTER (Chem. Abstr.).

Ether Convulsions. (*Brit. Journ. Anæsthesia, vol. viii, pp.* 142–7, 1936.) *Hoseason, A. S.*

Convulsions are associated with decrease in blood carbon dioxide, a tendency toward alkalosis and disturbances in the distribution of calcium. Intravenous injection of 10 c.c. of 10% calcium gluconate was useful as a preventive.

JAMES C. MUNCH (Chem. Abstr.).

Relation between Chemical Constitution and Pharmacological Actions of Convulsant Drugs. (*Sei-i-kai Med. Journ., vol. liv, No.* 12, *pp.* 2321–503 [*German abstr.* 7–10], 1935.) *Takeuchi, Shogoro.*

With frogs (*Rana nigromaculata*) and spinal frogs, 2-hydromorphine was less convulsant than morphine; 2-hydronitrosomorphine weaker than nitrosomorphine. On the other hand, 2-hydromorphine, nitromorphine, aminomorphine, codeine, nitrosocodeine, aminocodeine, thebaine and 2-hydrothebaine had the power to produce clonic spasm in mice, showing that an introduction of H_2, NO, NH_2 or CH_3 to the morphine mol. gives a new property which morphine itself does not have.

S. TASHIRO (Chem. Abst.).

The Action of Some Stimulants on Spinal Reflexes. (*Arch. Intern. Pharmacodynamic, vol. lii, pp.* 48–53, 1935.) *Knoefel, P. K., and Murrell, F. C.*

The action of caffeine, coramine and metrazole on spinal reflexes is similar to that of strychnine. In sufficient doses these drugs produce co-contraction of the flexor and extensor muscles after stimulation of the sensory nerve. The action of strychnine is strongest, with caffeine, metrazole and coramine in decreasing series.

M. L. C. BERNHEIM (Chem. Abstr.).

An Analysis of the Stimulant-Depressant Action of Caffeine, Coramine and Metrazole (Cardiazole) Based on Experimental Data. (*Arch. Intern. Pharmacodynamic, vol. lii, pp.* 373–80, 1936.) *Maloney, A. H.*

Caffeine, caffeine citrate, coramine and metrazole in certain concentrations temporarily antagonize the depressant action of the barbiturates on rats and rabbits. Higher concentrations of these drugs cause convulsions, delayed recovery or death. Divided doses gave no better results than single doses. Caffeine was much more effective than caffeine citrate.

M. L. C. BERNHEIM (Chem. Abstr.).

The Effect of Adrenaline, Anæmia and Carbon Dioxide on the Vasomotor Centre. (*Arch. Intern. Pharmacodynamic, vol. li, pp.* 463–87, 1935.) *Nowak, S. J. G., and Samaan, A.*

Adrenaline exerts no direct central vasodilator effect. The slight effects obtained can be reproduced in head perfusion experiments by increasing the perfusion pressure. After section of the cardio-aortic and carotid sinus nerves in

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the dog, anæmia has no effect until it is very extreme, when it causes a sudden increase in vasomotor tone. A reflex vasomotor stimulation occurs when the head, connected only by the spinal cord, is perfused with hyperapnœic blood.

M. L. C. BERNHEIM (Chem. Abstr.).

The Treatment of Epilepsy with Synergistic Calcium Bromide Therapy. (*Med. Press and Circ., vol. cxciii, pp.* 5–9, 32–6, 1936.) *Moore, M. T.*

Ten cases treated with calcium bromide are reported. In the majority of cases it appears superior to phenobarbital as regards control of seizures, influence on mental and emotional status and exudative manifestations.

P. D. ADAMS (Chem. Abstr.).

Delayed Results of the Malaria Therapy of Neurosyphilis. (*Sov. Psychonevr., No.* 3, 1936.) *Tarsis, F. I., and Landesman, J. U.*

The authors followed up the results of malaria therapy in 53 patients (41 males and 12 women) among 102 patients suffering from neurosyphilis that had received malaria treatment during the period between 1924 and 1927. They observed :

(1) Complete remission of 6–10 years' duration in 10 cases.
(2) Incomplete remission of 2½ to 3 years' duration in 7 cases.
(3) Improvement with arrest of the disease during 6–8 years in 7 cases.
(4) No effect at all in 13 cases.

Thus, a therapeutic effect after the malaria therapy of neurosyphilis was seen in 74·8% of cases.

The work is illustrated by several clinical cases. (Authors' Abstr.).

Alkali Reserve in Progressive General Paralysis Before and After Malaria Therapy. (*Bull. Acad. Med., vol. cxiv, pp.* 436–40, 1935.) *Paulian, D., and Tanasesco, G.*

The alkali reserve previous to treatment is normal in the blood ; the spinal fluid shows acidosis. After treatment the blood shows acidosis, and the spinal fluid is closer to normal but still slightly acid. A. E. MEYER (Chem Abstr.).

Treatment of Dementia Paralytica. (*Journ. Amer. Med. Assoc., vol. cvii, p.* 1031. *Sept.* 26, 1936.) *Barnacle, C. H., Ebaugh, F. G., and Ewalt, J. R.*

The authors report on the treatment of 60 patients, 30 by means of artificial fever and tryparsamide and 30 with malaria. Both groups were followed up with tryparsamide and bismuth.

The production of fever was carried out by means of the Kettering hypertherm, and the average time given was 44·5 hours of from 105° to 106° F. in ten equal sessions, with intervals of 4 to 7 days. Tryparsamide 2 grm. was administered at the height of each session, and following this course the patients received 3 grm. of tryparsamide at weekly intervals. In the malaria cases the average number of elevations of temperature was 9·9 and of chills 8·2. Each course was followed by six bi-weekly doses of neoarsphenamine before tryparsamide treatment.

Definite improvement occurred in 70% of the artificial fever series as compared with 63·3% of the malaria-treated cases. No patients died during the course of hyperpyrexia, but one died of cerebral hæmorrhage 2 weeks after completion of a course. One patient died during a course of malaria and 3 have died subsequently. The serological reactions of the cerebro-spinal fluid in both groups did not parallel the clinical results.

Of the artificial fever group the average age of the patients was 37·9 years and the average duration of symptoms 16·7 months, as compared with 42·6 years and 20·2 months respectively in the malaria group. Fifty patients had received no antisyphilitic therapy during the year prior to treatment. Of the remaining 10, 6 were in the fever group and 4 had malaria. T. E. BURROWS.

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The Treatment of Chorea Minor by Means of Electro-pyrexia. (*Journ. Amer. Med. Assoc., vol. cvii, p.* 938, *Sept.* 19, 1936.) *Neymann, C. A., Blatt, M. L. and Osborne, S. L.*

Twenty-five cases of chorea were treated by artificial fever produced by electro-magnetic induction. The average period of treatment and hospitalization was 16 days, an average of four treatments being given to each patient. In all cases the choreiform movements responded to treatment, but the chorea recurred in three cases.

Artificial fever carefully administered did not harm patients with rheumatic carditis, and clinical improvement was noted in all the patients with this condition. The temperatures employed are between 103° and 105° F. for a maximum of 8 hours. Some children develop convulsions with a fever over 105° F.

A decrease in respiratory rate to less than 12 per minute, cyanosis, an increase of the heart-rate to 160 per minute or complaints of shortness of breath or pain round the heart are indications for terminating the session. T. E. BURROWS.

Fever Therapy in Tabes Dorsalis. (*Journ. Amer. Med. Assoc., vol. cvii, p.* 845, *Sept.* 12, 1936.) *Bennett, A. E.*

The author reports on the treatment of 23 cases of tabes dorsalis and 1 case of spinal meningo-myelitis by means of the Kettering hypertherm combined with chemotherapy. Of the 14 patients who completed treatment, all but one had been having gastric crises or lightning pains or both when treatment began. The duration of symptoms varied from 1 to 14 years, and 7 patients had suffered for 5 years or longer. Eight of the cases were well advanced and 5 were incapacitated at least half the time.

The majority had had previous routine chemo-therapy, and several had had additional special forms of symptomatic treatment without success.

Nine of the cases received 50 hours or more of artificial fever above 105° F., and 5 cases received 30 hours, with treatments discontinued on account of complete relief of symptoms. Twelve patients have remained free from gastric crises and pains from 3 months to 1 year.

Various other symptoms which were also improved include ataxia, cord bladder, headaches, paræsthesia, trigeminal neuralgia and vertigo. No unusual complications during treatment were observed, but care must be taken to watch for hyperpyrexia and cerebral œdema during treatment. T. E. BURROWS.

Song in Aphasic Subjects [*Il canto negli afasici, i riflessi condizionati e la rieducazione del linguaggio*]. (*Riv. di Neurol., vol. ix, p.* 217, *June*, 1936.) *Baldi, F.*

It was found possible in two subjects to re-educate speech to some extent by getting the patients at first to imitate various sounds and songs. The author suggested that the process was one of establishing conditioned reflexes.

H. W. EDDISON.

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6. Pathology and Biochemistry.

The Particularly High Vitamin C Content of the Cerebro-spinal Fluid of Newborn Normal and Premature Infants. (*Compt. Rend. Soc. Biol., vol. cxxi, pp.* 987–8, 1936.) *Rohmer, P., Bezssonoff, N., and Stoerr, E.*

The spinal fluid of 5 normal full-term infants, 4–16 days old, contained 13–16 mgrm. ascorbic acid per litre, and that of 6 premature infants, 2–30 days old, contained 18–36, average 27, mgrm. per litre. The spinal fluid of normal infants 3 weeks to 5 months old contained 5–8 mgrm. per litre (same as adults).

L. E. GILSON (Chem. Abstr.).

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The Nature of the Reducing Substance of the Brain. I. Chemical Tests. (*Boll. Soc. Ital. Biol. Sper., vol. xi, pp.* 92–4; *Klin. Wochenschr., vol. xv, pp.* 483–4, 1936.) *Bonsignore, A.*

Brain-tissue contains substances which are capable of binding ascorbic acid in such a way as to modify its solubility in acetone. Extraction with an acid medium restores the solubility of ascorbic acid in acetone. On neutralization of the acid extract a precipitate forms which carries with it the reducing substance.

Conclusion: The reducing substance in the brain-tissue titratable by methods used by determining ascorbic acid is in fact ascorbic acid.

Ibid. II. Biological Tests. (*Boll. Soc. Ital. Biol. Sper., vol. xi, pp.* 94–6; *Klin. Wochenschr., vol. xv, pp.* 742–3, 1936.) *Bonsignore, A., and Ardy, C.*

The administration of fresh brain-tissue in amounts corresponding to 0·50 mgrm. of reducing substance expressed as ascorbic acid prolonged markedly the life of guinea-pigs maintained on a scorbutic diet. This indicates that all or a large part of the reducing substance in brain-tissue is ascorbic acid.

PETER MASUCCI (Chem. Abstr.).

Variations in the Dehydroascorbic Acid-Ascorbic Acid Ratio in the Heart after the Administration of Parasympatheticotropic Substances. (*Boll. Soc. Ital. Biol. Sper., vol. xi, pp.* 75–7, 1936.) *Copello, F.*

Drugs which excite or paralyse the parasympathetic system exert a marked action on the dehydroascorbic acid-ascorbic acid ratio of the heart of the guinea-pig; the exciting drugs decrease this ratio, while the paralysing drugs increase it. The reducing power of the heart-tissue is increased by exciting the parasympathetic system and decreased by paralysing it. PETER MASUCCI (Chem. Abstr.).

The Ascorbic Acid Content of Various Parts of the Central Nervous System and of the Peripheral Nerves. (*Arch. Ges. Physiol.* [*Pflüger's*], *vol. ccxxxvii, pp.* 216–21, 1936.) *Melka, J.*

The ascorbic acid contents of the cerebellar cortex, cerebral cortex, globus pallidus and corpus callosum are 0·26, 0·17, 0·16 and 0·13 mgrm. per grm. respectively. The peripheral nerves contain about 0·03 mgrm. per grm. of tissue.

A. G. (Chem. Abstr.).

Vitamin C Content of Different Parts of the Nervous System. (*Zeitschr. f. d. ges. Neur. u. Psychiat., vol. cliii, pp.* 182–92, 1935.) *Plaut, F., and Bülow, M.*

The ascorbic acid content of all parts of the human brain declined with advancing age, but at relatively different rates. The cerebral content was greatest in the fœtus, the cerebellar content in infancy, and that of the spinal cord in old age. The content of the cerebral and cerebellar cortex was greater than that of the medulla at all ages except in senility. Observations on the rabbit, dog, cat and calf gave comparable results. B. C. A. (Chem. Abstr.).

Vitamin C in Spinal Fluid in Normal and Pathological Conditions. (*Bull. Acad. Med., vol. cxiv, pp.* 803–10, 1935.) *Marinesco, G., Buttu, G., and Olteanu.*

The vitamin C in spinal fluid decreases consistently during life from 2·2–2·8 mgrm. before 10 years of age to 0·2–0·3 mgrm. per 100 c.c. after 70 years. Changes occurring in various diseases of the central nervous system are reported.

A. E. MEYER (Chem. Abstr.).

Potassium in the Brain in Vitamin B_1 Deficiency. (*Nature, vol. cxxxviii, p.* 308, 1936.) *Kinnersley, H. W.*

No difference was found between the amounts of potassium in the brains of normal and vitamin B_1 deficient pigeons. The values were 3·6 mgrm. per grm. of tissue in each case. The changes obtained by Ballif and Gherscovici cannot therefore, have been due to the avitaminosis. E. D. WALTER (Chem. Abstr.).

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Lesions of the Nervous System in Vitamin Deficiency. IV. The Effect of Carotene in the Treatment of the Nervous Disorder in Rats Fed on a Diet Low in Vitamin A. (*Journ. Nutrition, vol. xi, pp.* 411–23, 1936.) *Zimmerman, H. M., and Cowgill, G. R.*

Albino rats subsisting on an artificial ration adequate in all dietary essentials except vitamin A develop a nervous disorder which is characterized by muscular weakness, inco-ordination and, in the severest cases, paralysis of the posterior extremities. There is a demyelinization of the peripheral nerves. Carotene, when administered late in the course of this dietary deficiency or in inadequate dosage (50 microg.), does not alter the fatal outcome in any way. When administered early in the course of the deficiency in adequate amounts (100 microg.), carotene is effective in restoring rats to good health, the neurological disorder alone persisting. Animals thus treated with carotene resume their normal rate of growth and recover from xerophthalmia, but the anatomical changes in their nervous system are even more marked than in the untreated animals. This is attributed to the relative inability of the nervous system to regenerate when once severely injured. There is no evidence of this deficiency when the same artificial ration is supplemented with 50 microg. daily of carotene. This study shows that the changes in the nervous systems of animals fed on the artificial diet low in vitamin A are due to a deficiency of this factor and not to a deficiency of an unsaturated fatty acid.

C. R. Fellers (Chem. Abstr.).

The Prevention of Nutritional Encephalomalacia in Chicks by Vegetable Oils and Their Fractions. (*Journ. Biol. Chem., vol. cxiv, pp.* 673–87, 1936.) *Goettsch, M., and Pappenheimer, A. M.*

Certain edible oils (corn oil, cottonseed oil, hydrogenated cottonseed oil, peanut oil and soybean oil) when added to a causative diet provide a protective factor against nutritional encephalomalacia of chicks. This disease is not thought to be a manifestation of vitamin E deficiency, since the addition of natural food-stuffs rich in vitamin E does not protect the chicks. Cabbage, alfalfa-leaf meal, meat scraps and fresh beef liver exert no beneficial effect, while grains and grain products, lettuce, spinach, grass, egg yolk and calf brain provide only partial protection or yield inconstant results. On diets low in lard content the incidence is decreased; however, with complete elimination of lard from the diet the disease still appeared. The protective factor, found in the edible oils, can be partially extracted by 95% alcohol; it is thermostable, resistant to aëration and ultraviolet irradiation, and is present in the non-saponifiable fraction of the oil if due precaution is taken against oxidation during saponification. The fatty acid and water-soluble fractions are inert.

H. G. Shurecht (Chem. Abstr.).

Lesions of the Central and Peripheral Nervous Systems Produced in Young Rabbits by Vitamin A Deficiency and High Cereal Intake. (*Brain, vol. lviii, pp.* 141–73, 1935.) (*Physiol. Abstracts, vol. xx, pp.* 699–700.) *Mellanby, E.*

Rabbits 8–10 weeks old were fed on a diet compatible with general good health and growth, but deficient in carotene and vitamin A. This diet produced in all cases lesions in the peripheral and central nervous system from which the animals can be protected by the addition to this diet of pure carotene (1–3 mgrm. daily), of some source of vitamin A, or of cabbage or carrot. The bearing of these results upon the general question of the specific relation between nutritional substances and the structure and proper activity of the nervous system as a whole is discussed.

E. Bame (Chem. Abstr.).

The Presence of Spirochæta pallida in the Vegetative Nervous System [*Sulla Presenza della Spirocheta pallida nel Sistema Nervoso Vegetativo*]. (*Neopsichiatria, vol. ii, p.* 165, *March–April*, 1936.) *Benvenuti, M.*

Two cases of general paralysis are reported in which spirochætes were found in the vegetative nervous system, both in the ganglia of the lateral sympathetic and in the perivisceral ganglia.

H. W. Eddison.

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The Blood Bromine in Manic-depressive Psychoses [*Bromo Ematico Nella Frenosi Maniaco-depressiva*]. (*Riv. Sper. di Freniat., vol. lx, p.* 185, *June,* 1936.) *Bergonzi, M.*

After the examination of a series of 32 cases the author concludes that the bromine content of the blood is low in cases of manic-depressive psychosis untreated with bromides, the administration of which increases the figure in proportion to the doses administered. H. W. EDDISON.

Relations between Acetylcholine, the Vagal Substance of Loewi and Viale's Vagotropin. (*Boll. Soc. Ital. Biol. Sper., vol. xi, pp.* 88–91, 1936.) *Bartolini, B.*

Vagotropin differs from acetylcholine in the following characteristics: it is not extracted by ether; it does not act on the dorsal muscle of the leech; it remains unaltered in the serum; it is precipitated by CCl_3CO_2H; it is not influenced in its action by eserine. The vagal substance of Loewi is either identical with acetylcholine or possesses properties similar to acetylcholine.

PETER MASUCCI (Chem. Abstr.).

Studies on the Origin of Viale's Vagotropin. (*Boll. Soc. Ital. Biol. Sper., vol. xi, pp.* 83–5, 1936.) *Bartolini, B.*

Serum was drawn from the jugular, hepatic, portal, pancreatic, splenic, femoral and suprarenal veins. Their vagotropic action was tested on the intestine. The results do not justify the conclusion that vagotropin is present in larger amounts in some parts of the organism than in others.

PETER MASUCCI (Chem. Abstr.).

The Nature of Viale's Vagotropin. (*Boll. Soc. Ital. Biol. Sper., vol. xi, pp.* 86–8, 1936.) *Bartolini, B.*

Vagotropin is an organic substance, as it is not present in the ash of serum. It is alcohol-soluble, but is not extracted from serum either by ether or chloroform. The substance is not present in either the trichloracetic acid or phosphotungstic acid filtrates. Vagotropin is not precipitated by tannic acid.

PETER MASUCCI (Chem. Abstr.).

The Pathology of Tuberculous Meningitis: II. Tissue Chemistry. (*Monats. f. Kinderheilk., vol. lxiv, pp.* 263–7, 1935–6; *cf. C.A., vol. xxix, Col.* 8112.) *Moritz, Denes v., and Kulcsar, Margarete.*

The brains of 10 infants and children dead of tuberculous meningitis were analysed for Cl, residual N, total solids and effect on the f.p. Cl is decreased, significantly more in grey than in white matter, the residual N is normal or lowered, and the f.p. is higher, as compared with normal brains. It is probable that hypochloræmia and hypo-osmosis contribute to the nervous symptoms.

E. M. HUMPHREYS (Chem. Abstr.).

Influence of Bile-salts on the Nervous System Following Intraspinal Usage. (*Proc. Soc. Exptl. Biol. Med., vol. xxxii, pp.* 1201–4, 1935.) *Lichtman, S. S., and Stern, E. L.*

In the cat Na desoxycholate in minute doses in aqueous solution was injected intra-spinally in the lumbar region without untoward effects or injury to the cord; larger doses produced motor and sensory disturbances and even death from respiratory paralysis. Traumatized spinal cord tissue was highly susceptible to alcohol solutions of bile-salts. Spinal-fluid protein and cord tissue reduced the hæmolytic action of bile-salts. C. V. BAILEY (Chem. Abstr.).

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Psychosin Sulphate: Its Action upon Bacteria, Toxins, Serum and Red Cells. (*Journ. Path. Bact., vol. xlii, pp.* 363–98, 1936.) *Drury, A. N., et Al.*

Psychosin, sphingosin, ethyl psychosin and methyl sphingosin derived from cerebrosides obtained from fresh brain have definite biological activities. They all kill bacteria, hæmolyse red cells, and when mixed with serum lead to characteristic precipitates. Psychosin alone can "fix" toxins, and its action is much more potent on tetanus than on diphtheria toxin. Deaminized psychosin possesses none of these activities. There is no evidence that any of these substances exists in the body as such, but psychosin and sphingosin are constituents of substances found throughout the body. Surface adsorption may be related to the serum precipitates and the bactericidal action. JOHN T. MYERS (Chem. Abstr.).

Conceptions and New Researches on the Ætiology and Pathogenesis of Cerebral Hæmorrhage: I. The Role of Certain Products of Cellular Metabolism: Critical and Experimental Study. (*Journ. Physiol. Path. Gen., vol. xxxiii, pp.* 64–74, 1935.) *Frommel, E., and Herschberg, A. D.*

The intracerebral injection in guinea-pigs of cerebral extracts, adenosine-phosphoric acid, histidine, histamine and tryptophane causes hæmorrhage, while glycocoll, tyrosine and leucine do not, but act as vaso-constrictors. On the inter-digital membrane of the frog and the mesentery of the toad adenosine-phosphoric acid, histamine and histidine are active vaso-dilators, glycocoll is a feeble dilator, while tryptophane, leucine and tyrosine constrict. These observations are considered to support the view that cerebral hæmorrhages are due to local metabolic disturbances. L. A. MAYNARD (Chem. Abstr.).

A New Hapten in the Protagon Fraction of Brain. (*Z. Immunitäts., vol. lxxxvii, pp.* 426–44, 1936.) *Schwab, E.*

That part of brain which is insoluble in petroleum ether and soluble in hot alcohol, and which corresponds in properties and composition to protagon, gives a reaction with brain antiserum, and produces by combination immunization anti-bodies which react with protagon but not with the usual alcohol extract of brain. Brain-suspension antisera, but not brain-extract antisera, and protagon antisera react with the protagon. Apparently there are two haptens in brain, one soluble in cold alcohol and the other only in hot alcohol. The often observed anti-complementary action of protagon solutions is prevented by the addition of lecithin, but such mixtures will sometimes react with brain-extract antisera although the individual components do not. The distinction of the protagon hapten from the already known brain hapten can further be shown by cooling the hot alcohol extract of brain. The protagon anti-serum reacts with the sediment but not with the mother liquor. The two haptens can also be demonstrated by adsorption experiments. Protagon is not found in parenchymatous organs, but does occur in all parts of the central nervous system and in the posterior pituitary. Antisera for white brain matter reacts more strongly with protagon than does that for grey brain matter. JULIAN H. LEWIS (Chem. Abstr.).

Iso-electric Points of the Blood-serum of the Insane. (*Arch. Phys. Biol., vol. xiii, pp.* 56–9, 1936.) *Tomioka, T.*

Samples from 17 male schizophrenics were examined. The iso-electric points were abnormal, indicating some change in the blood proteins, but the results were too irregular to permit of definite conclusions. L. E. GILSON (Chem. Abstr.).

The Glycerophosphatase of the Brain. (*Boll. Soc. Ital. Biol. Sper., vol. x, pp.* 374–6, 1935.) *Cedrangolo, F.*

The maximum of catalytic activity in the rabbit and guinea-pig brain is at a pH 5·32 and 9·16, and at a concentration of glycerophosphate 0·0016 *M*. Enzyme

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extracts can be purified by iso-electric precipitation with 0·1 *N* HCl. Inactivated extracts are reactivated by Mg^{++}. Phlorizin, NaF, iodoacetic acid and As_2O_3 have an inhibiting effect. The *α*-acid is split more easily than the *β*-acid.

A. E. MEYER (Chem. Abstr.).

Mitochondria, Golgi Apparatus and Vitamins. (*Australian Journ. Exptl. Biol. Med. Sci., vol. xiii, pp.* 239–49, 1935.) *Bourne, G.*

Using the acid-$AgNO_3$ technique for vitamin C, the Carr-Price reaction for vitamin A and the nitro-prusside reaction for glutathione, Bourne found evidence that mitochondria have an outer lipoid cortex containing vitamin A and generally vitamin C, and an inner aqueous phase rich in vitamin C and glutathione. In scurvy, vitamin C is first withdrawn from the Golgi apparatus and then from the mitochondria to very low values. The vitamins and glutathione are thought to function in the respiration of the mitochondria. There was no evidence of vitamin C being present in the oxidized form. C. G. KING (Chem. Abstr.).

Central Regulation of Fat Metabolism in the Epidermis. (*Arch. Derm. and Syph., vol. clxx, pp.* 511–20, 1934.) *Perutz, A., Lustig, B., and Klein, A. E.*

Destruction of the midbrain followed by prolonged feeding with fatty substances in rabbits increases hair fat and blood *β*-hydroxybutyric acid, blood ketones being unaffected. Fat excretion by the skin is therefore probably regulated by a central mechanism. B. C. A. (Chem. Abstr.).

Experimental Epilepsy. III. The Change in the Protective Qualities of the Serum in the Case of Epileptic Fits Produced by Camphor. (*Kolloid Z., vol. lxxiv, pp.* 218–21, 1936.) *Nekludov, V.*

A modification of the Rubinzahl method is well adapted to determining the protective ability of colloids. When the epileptic fits were violent, often killing the experimental animal, the protective ability of the serum diminished during the course of the experiment. If, after the fits, complete healing of the animal took place, the protective capacity of the serum increased.

W. G. PARKS (Chem. Abstr.).

The Role of Calcium in Spontaneous Overbreathing Tetany. (*Quart. Journ. Med., vol. v, pp.* 341–54, 1936.) *Barnes, C. G., and Greaves, R. I. N.*

In the three cases studied there was an alkalosis, as shown by increased alkaline reserve and increased pH of the urine. This may be responsible for the decrease in diffusible calcium of the blood. During tetany there was a definite drop in the diffusible calcium of the spinal fluid (5·0 to 4·5 mgrm. %), which is an index of the diffusible calcium in the blood. J. T. MYERS (Chem. Abstr.).

The Dielectric Behaviour of Organic Zwitter Ions, with Special Reference to Molecules of Brain and Nerve Substance. (*Sitzber. Heidelberg. Akad. Wiss., Math.-Naturw. Klasse, No.* 6, *p.* 41, 1935.) *Hausser, I.*

A short-wave apparatus is described, with drawings and photographs, for measurement of the dielectric constituent ε of solutions $\Delta\varepsilon/\Delta c$ (c = concentration). Values are given for some amino-acids and their betaines and for cephalin, lecithin, sphingomyelin and cerebron. The solvents are water and alcohols. The betaines show anomalous dispersion in alcohol solution and therefore exist as zwitter ions.. Lecithin and sphingomyelin show anomalous dispersion in alcohol solution at a wave-length of about 4 m. owing to the two long, weakly polar arms exerting a directing force on the short, strongly polar arm (cholinephosphoric acid), giving it a pendulum-like oscillation. The bearing of these results on the possible explanation of electric processes in brain and nerve substance is suggested.

E. D. WALTER (Chem. Abstr.).

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Neuro-central Regulation of Blood Nitrogen. (*Boll. Soc. Ital. Biol. Sper., vol. x, pp.* 225–7, 1935.) *Jedlowski, P.*

Blood nitrogen is increased by destruction of one or both of the hemispherical cortices in the rabbit, and to a small extent by lesions of different parts of the encephalic base. B. C. A. (Chem. Abstr.).

The Biochemistry of the Nervous System during Pregnancy. (*Ukrain. Biochem. Journ., vol. ix, pp.* 55–67, 1936.) *Rashba, O. Ya.*

There is no change in the creatine and water contents and only a slight decrease, if any, in the total nitrogen content of the brains of rabbits or of guinea-pigs during the second half of pregnancy. The creatine content of the brain is subject to seasonal variations. E. E. Stefanowsky (Chem. Abstr.).

Hypoglycæmia and Convulsions in Pigeons: I. (*Riv. Patol. Sper., vol. xii, pp.* 105–26, 1934.) *Moracci, E.*

It is confirmed that insulin convulsions appear readily in starved pigeons, though normal ones can tolerate large doses. If the starved birds have also been kept short of water, convulsions appear less readily, probably because of the high blood-sugar levels reached under these conditions. There is, however, no particular blood-sugar level at which convulsions appear; in fact, they may do so while the value is still relatively high. E. Bame (Chem. Abstr.).

Acetylcholine in Traumatic Epilepsy. (*Presse Méd., vol. xliii, pp.* 1892–5, 1935.) *Fribourg-Blanc, Lasalle and Passa.*

Favourable results are reported. They are attributed to relief of vascular spasms. A. E. Meyer (Chem. Abstr.).

Glycolysis and Phosphate Exchange in Cell-free Brain Extracts of Normal Mammals. (*Zeitschr. Physiol. Chem., vol. ccxl, pp.* 265–78, 1936.) *Euler, H. v., Günther, G., and Vestin, R.*

The experiments were designed to determine whether the breakdown of carbohydrate in the mammalian brain occurs in a fundamentally different manner from that demonstrated for muscle tissue, i.e., whether the breakdown takes place without intervention of phosphate esters. Using cell-free brain extracts of calf, rabbit and rat, experiments on the removal of phosphate from extracts having glycolytic activity, either by dialysis or by calcium precipitation, showed that the breakdown required the presence of phosphate. This phosphate requirement is, however, smaller than for muscle glycolysis. In the extract of calf brain there occurs in the presence of NaF a reaction of acetic acid with hexosediphosphoric acid to form lactic acid and a difficultly dialysable ester (phosphoglyceric acid), as is also the case with muscle extract. The fact that this intermediate reaction of muscle glycolysis occurs also in brain glycolysis warrants the conclusion that phosphoric esters participate also as intermediates in brain glycolysis. Cozymase (active or heat inactivated), adenylic acid and adenosinetriphosphoric acid, which function in the rephosphorylation in muscle glycolysis, are active also in brain glycolysis. Substrates of brain glycolysis are glucose and fructose, and less effectively glycogen. Glutathione alone cannot activate the enzyme system of brain glycolysis without the addition of phosphate transporters such as cozymase and adenosinetriphosphate. A. W. Dox (Chem. Abstr.).

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Calcium and Protein Changes in Serum During Sleep and Rest Without Sleep. (*Amer. Journ. Physiol., vol. cxvi, pp.* 531–4, 1936.) *Cooperman, N. R.*

During sleep (5–7 hours) both total serum calcium and serum proteins decrease, with a resultant increase in the calcium-ion concentration of the serum. During rest without sleep for the same period similar changes occur. In shorter periods of sleep and rest there is a more marked decrease in total serum calcium, but the ionized calcium remains the same. The changes found in the shorter periods of sleep and rest can be correlated with an increase in circulating plasma volume.

E. D. W. (Chem. Abstr.).

Carbohydrate Metabolism in Mental Disorders. I. Glucose-tolerance Tests in Manic-depressive Insanity and Other Depressions. (*Edinburgh Med. Journ., vol. xliii, pp.* 524–7, 1936.) *Tod, H.*

The work of McCowan and Quastel was confirmed in cases of depression and mania, but in cases of stupor higher hyperglycæmic indexes were found.

R. Brown (Chem. Abstr.).

Chemical Transmission of Nervous Influx through Central Synapses. (*Compt. Rend. Soc. Biol., vol. cxxii, pp.* 1128–32, 1936.) *Benetato, G., and Munteanu, N.*

A dog was anæsthetized with amytal and ether and treated with eserine. Efferent blood from the medulla was re-injected into the carotid artery. There was no effect on respiration until the central end of the vagus was electrically stimulated, then the re-injected blood caused a slowing of respiration, indicating the presence of acetylcholine. The leech-muscle reaction for acetylcholine was also obtained with blood collected during stimulation of the vagus.

L. E. Gilson (Chem. Abstr.).

The Cerebral Origin of Cholesterolæmia. (*Therap. Arch.* [*U.S.S.R.*], *vol. xiii, pp.* 132–9, 1935.) *Snachkova, A. A.*

In cholesterolæmia, which appears in dogs and cats under the influence of deficient kidney functioning or of electric stimulation of the brain, a proportionately larger amount of cholesterol is observed in the blood from the brain (V. jugularis) than in the blood of the V. femoralis. Likewise, the cholesterol content of the blood flowing from the kidneys is lowered. Cholesterolæmia develops, therefore, as a result of a mobilization of the cholesterol depot, the cerebral substance. It is of clinical importance that this be recognized, as it indicates certain trauma of the central nervous system. M. G. Moore (Chem. Abstr.).

Recent Advances in Knowledge Concerning the Chemical Mediation of Nerve Impulses. (*Proc. Staff Meetings Mayo Clinic, vol. xi, pp.* 327–31, 1936.) *Butt, H. R.*

Studies recently reported in the literature show that sympathin, a compound related to adrenaline, exists in two forms—sympathin E, given off by smooth muscle which contracts on sympathetic stimulation, and sympathin I, given off by smooth muscle which is inhibited or relaxed on sympathetic stimulation. It has been shown that these substances may enter the blood-stream, and thereby produce similar effects on smooth muscle elsewhere than where the original contraction took place. Peripheral parasympathetic effects are transmitted by the liberation of an acetylcholine-like substance which is rapidly destroyed in circulating blood by an esterase; this destruction can be prevented by the use of physostigmine. The cholinesterase is of an enzymic nature, and has been established as a specific destructive agent for acetylcholine. Unconfirmed evidence intimates that stimulation of nerves to voluntary, striated muscle-fibres produces acetylcholine. There is also evidence that acetylcholine is liberated at ganglionic synapses. Substantive evidence indicates that the chemical receptive action of a tissue is attributable to the type of nerve-fibre that supplies it. R. C. Wilson (Chem. Abstr.).

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The Antigenic Properties of Lecithin and Cephalin. (*Journ. Immunol., vol. xxvi, pp.* 25–48, 1934.) *Wadsworth, A., Maltaner, E., and Maltaner, F.*

Purified preparations of cephalin (from beef brain) and lecithin (from beef brain, liver and heart) were tested for antigenic power in complement-fixation tests and when injected into the animal body. These phosphatides, dispersed by several methods, were inactive with or without added cholesterol, when used as the antigen in complement-fixation tests with syphilitic sera. When mixed with pig-serum and injected into rabbits, the purified phosphatides produced no antibodies (complement-fixation tests) to the purified or compound phosphatide or to beef-heart extracts. No evidence could thus be obtained that these compounds are antigenic in the tissues. Compound preparations of the phosphatides, mixed with pig-serum and injected into rabbits, produced antisera which would fix complement in conjunction with the compound phosphatides or with Wassermann antigens, but not with the purified phosphatides. The results suggest that the specific antigenic activity of the compound preparations is due to some contaminating substance. Compound lecithin and cephalin adsorbed on collodion particles were not antigenic when injected into rabbits. E. BAME (Chem. Abstr.).

Spontaneous Creatinuria in Nervous Diseases. (*Deutsch. Arch. f. Klin. Med., vol. clxxviii, pp.* 387–95, 1936.) *Kostakow, St.*

Metabolic studies on 50 subjects show that a spontaneous creatinuria is frequently found in nervous affections both with and without muscular atrophy; apparently no single factor can account for its appearance in diseases of widely different origins. Certain distinctions can be made, however, among creatinuric subjects; administration of glycine to myopathic subjects results in an immediate increase in creatine elimination; other subjects (except in the regenerative stages of poliomyelitis) show no such variation after treatment with glycine. Apparently the variations in the amounts of creatine eliminated depend not alone upon a disturbance of creatine metabolism, but upon other metabolic functions as affected by the diseased muscles. Except in neural or spinal atrophy, or in poliomyelitis, subjects react normally toward creatine, given in doses of 3 gr., in spite of disturbances in endogenous creatine-creatinine metabolism. No spontaneous creatinuria is observed in dystrophia adiposo-genitalis, brain tumour or hyperthyroid conditions. P. Y. JACKSON (Chem. Abstr.).

Effect of Dichlorodiethylsulfone on Brain Respiration. (*Nature, vol. cxxxviii, pp.* 327–8, 1936.) *Peters, R. A.*

Iodoacetic acid and D.E.S. (dichlorodiethylsulfone) have a similar effect upon the pyruvate oxidase of the brain respiration system *in vitro*.
F. SAUNDERS (Chem. Abstr.).

Physicochemical Processes in Nerve Tissue. I. Oxidation-reduction Potentials of the Brain of the Rabbit in Cases of Poisoning by Strychnine and Cocaine. (*Ukrain. Biochem. Journ., vol. ix, pp.* 43–53, 1936.) *Fomin, S. V., and Strashesko, D. N.*

In cases of poisoning by strychnine and cocaine in aërobic and anaërobic conditions, the potentials establish themselves quicker (in 60 minutes) as compared with the normal rate. Potentials of boiled extracts of the brain-tissue of strychnine-poisoned rabbits are lower than those of similar extracts of a normal tissue. In anaërobic conditions these potentials are considerably higher than normal both in boiled and unboiled samples. In cases of poisoning by cocaine in aërobic conditions the potentials of extracts of both boiled and unboiled samples are considerably lower during the first two hours; in boiled samples it afterwards approximates

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the normal. An abrupt deviation of potentials to the negative side in the unboiled samples should be due to the strong reducing properties of extracts of brain-tissue. In anaërobic conditions the differences of potential in normal and poisoned conditions are lower. E. E. STEFANOWSKY (Chem. Abstr.).

The Action of the Nervous System on the Metabolism of Depot Lipides. (*Arch. Sci. Biol.* [*Italy*], *vol. xxi, pp.* 570–8, 1935.) *Cedrangolo, F.*

In rabbits fed normally, severing of the nerves supplying the interscapular adipose bodies determines an increase in fat preceded by an increase in glycogen. With reduced diets the section of nerves impeded the mobilization of fat from the denervated body. P. F. METILDI (Chem. Abstr.).

The Demonstratione of Tryptophane in the Cerebro-spinal Fluid in Tuberculous Meningitis. (*Monatsschr. f. Kinderheilk., vol. lxv, pp.* 41–4, 1936.) *Bock, Harald.*

In all 19 cases of tuberculous meningitis the cerebro-spinal fluid gave a positive colour test for tryptophane, while only 3 of 100 control cases reacted positively. This test is an aid to the early diagnosis of tuberculous meningitis. E. M. HUMPHREYS (Chem. Abstr.).

The Calcium Content of Cerebro-spinal Fluid in Experimental Tetany. (*Zeitschr. f. Kinderheilk., vol. lvii, pp.* 467–73, 1935.) *Siwe, Sture.*

The Ca content of cerebro-spinal fluid does not vary greatly from the ultrafiltrable Ca of the blood-serum, while the P is regularly lower than in the serum ultrafiltrate. When the Ca : P relationships of the serum are altered, as in phosphate tetany, a parallel shift does not occur, and osmotic relations in the fluid are maintained by a migration of other ions. This signifies that selective action rather than simple dialysis determines the composition of cerebro-spinal fluid. E. M. HUMPHREYS (Chem. Abstr.).

The Quinine Test : A New Spinal-fluid Reaction. (*Tôhoku Journ. Exptl. Med., vol. xxvii, pp.* 357–9, 1935.) *Takata, M.*

Normal spinal fluid showed no immediate reaction with 2% quinine. The fluid from certain pathological cases became turbid within 15 minutes or less as a result of precipitation of globulins and albumins by the quinine. The test will show the presence of 0·04% protein in the spinal fluid.
S. A. CORSON (Chem. Abstr.).

Crystallization of (*Sodium*) *Chloride in Presence of Cerebro-spinal Fluid.* (*Bull. Acad. Med. Roumanie, vol. i, pp.* 133–40, 1936.) *Romesco, P., Cosmulesco, I., and Serban, F.*

Sodium chloride in presence of cerebro-spinal fluid crystallizes in various abnormal forms dependent on the composition of the fluid ; the phenomenon is probably of diagnostic value. B. C. A. (Chem. Abstr.).

Glutathione of Normal and Pathological Cerebro-spinal fluid. (*Anales Farm Bioquim.* [*Buenos Aires*], *vol. vii, pp.* 11–15, 1936.) *Braier, B.*

The method of Langou and Marenzi was used. Spinal fluid from normal men contained 3·8–6·9, average 5·5 mgrm.%, total glutathione, and the average for six normal women was 4·86. Samples from men with various forms of insanity contained 7·1–11·2, average 9·25, where the globulin and Wassermann tests were positive, and 5·3–6·8, average 6·1, if these tests were negative.
L. E. GILSON (Chem. Abstr.).

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The Effect of Induced Hyperglycæmia on the Glucose Content of Cerebro-spinal Fluid. (*Quart. Journ. Med., vol. v, pp.* 169–85, 1936.) *Cohen, H., and Libman, J.*

If the sugar content of the blood is raised for a sufficient period, there is a significant rise in the sugar content of the spinal fluid.

J. T. MYERS (Chem. Abstr.).

The Glucose Content of Cerebro-spinal Fluid after Withdrawal. (*Quart. Journ. Med., vol. v, pp.* 183–5, 1936.) *Cohen, H., and Libman, J.*

The sugar content of spinal fluid kept at room temperature or at 37° remained constant up to 94 hours, but it fell rapidly if contaminated with micro-organisms, *B. subtilis* being most common. J. T. MYERS (Chem. Abstr.).

A Comparative Study of the Distribution of Electrolytes (Cl, P, Ca, Mg) in the Cerebro-spinal Fluids of Animals and Man. (*Arch. Sci. Biol.* [*U.S.S.R.*], *vol. xxxix, pp.* 209–17, 1935.) *Fridman, A. P., and Petrova, V. V.*

The following average figures in mgrm.% are reported, with minimum and maximum in parentheses: Chlorine (as sodium chloride)—man 730 (725–50), cattle 633 (611–713), dogs 667 (602–783); inorganic phosphorus—man 1·75 (1·5–2·1), cattle 3·20 (4·06–2·15), dogs 3·09 (2·82–3·47); calcium—man 6·2 (4·6–6·8), cattle 6·58 (6·01–7·18), dogs 6·56 (5·13–7·40); magnesium—man 2·7 (1·02–3·5), cattle 2·17 (2·13–3·20), dogs 3·09 (2·58–3·81). Some attempt is made to correlate the figures with age, state of nutrition, sex, etc., but the number of observations is insufficient for significant evaluation. W. A. PERLZWEIG (Chem. Abstr.).

The Cerebro-spinal Fluid Sugar in Animals and in Man. (*Arch. Sci. Biol.* [*U.S.S.R.*], *vol. xxxix, pp.* 539–46, 1935.) *Fridman, A. P., and Arkina, R. Ch.*

The results of 360 analyses of spinal-fluid sugar by the Hagedorn-Jensen method are reported. Normal human fluid contains 42·5–60·5 mgrm.%, average 49 mgrm.%. The cisternal fluid of cattle (cows and bulls) in adults averages 47 mgrm.% and calves 51 mgrm.%. In sheep and goats the average is 51 mgrm.%; in dogs 45–77 mgrm.%, average 64 mgrm.%. There do not appear to be any significant differences in the species tested. W. A. PERLZWEIG (Chem. Abstr.).

Reducing Substances in Human Cerebro-spinal Fluid. (*Boll. Soc. Ital. Biol. Sper., vol. xi, pp.* 332–4, 1936.) *Mitolo, M.*

The reducing effect of the fluid, in acid medium, on 2, 6-dichlorophenol-indophenol varied greatly in the 22 cases studied. From 0·75 to 4·57 c.c. fluid was required to oxidize 0·5 c.c. indicator. H. L. GRUEHL (Chem. Abstr.).

Protein Fractions in Normal and Pathological Cerebro-spinal Fluids in Childhood. (*Monatsschr. f. Kinderheilk., vol. lxvi, pp.* 137–84, 1936.) *Ujsaghy, P.*

Total proteins and protein fractions were determined on the cerebro-spinal fluids of 58 normal children and 122 children with diseases of the brain and meninges. The values for the fluids of the normal group, beyond the age of 3 months, were: total protein 18 to 26 mgrm.%, average 21 mgrm.%; albumin 13 to 21 mgrm.%, average 17 mgrm.%; globulin (pseudo-) 4 to 6·6 mgrm.%, average 5·3 mgrm.%. Euglobulin and fibrinogen were not found except in the fluids of young infants. Newborn premature infants in particular have high values. Detailed analyses are given for the changes in many pathological conditions. Especially striking are the increases, particularly of euglobulin and fibrinogen, in inflammatory and some non-inflammatory conditions, and the marked absolute increase of globulin in juvenile taboparesis. In some of these cases there is a reversal of the globulin: albumin ratio from 0·5 or below to 1·0 or more. E. M. HUMPHREYS (Chem. Abstr.).

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Sugars and Glycolytic Enzymes of Spinal Fluid in Epidemic Cerebro-spinal Meningitis. (*Journ. Clin. Invest., vol. xv, pp.* 519–23, 1936.) ***Hubbard, R. S., Russell, N. G., jun., and Russell, N. M.***

Specimens of spinal fluid from two patients with meningococcic meningitis gave a positive test for fructose. Incubation destroyed reducing substance but not fructose, the glycolytic enzyme responsible for this change being present in the leucocytes. J. B. BROWN (Chem. Abstr.).

Cerebro-spinal Fluid in Vascular Diseases of the Central Nervous System. (***Bull. Neur. Inst. New York, vol. v, p. 5, Aug.,*** 1936.) ***Brewer, E. D., and Hare, C. C.***

A series of 317 patients with vascular diseases of the nervous system were studied, and the results compared with those of spinal fluids from 186 patients with cerebral tumour. The spinal fluids associated with vascular diseases were normal in 55·6% of the cases as concerns total protein content and globulin reaction. The fluids associated with brain tumour were normal in 44·6%.

Fluids from patients with vascular disease of the spinal cord were not characteristic. An elevation of the spinal manometric pressure over 180 mm. of water definitely favoured the diagnosis of brain tumour, as did to a less degree the presence of more than 100 mgrm. per 100 c.c. of total protein.

A pleocytosis and xanthochromia occurred not infrequently in both conditions.

The vascular diseases occurred in the proportion of 68·6% males to 31·4% females. T. E. BURROWS.

ERRATA.

In Dr. A. Beck's article on "Immunity against *Spirochæta pallida*" in the May, 1936, number, at bottom of p. 254 the following correction should be made:

For "the statement of Truffi that spirochætes are able to survive for 5 hours in birds' blood, which has a normal temperature of 45° C.", read "the statement of Truffi that spirochætes are able to survive for 5 hours in bird's blood, *the normal temperature of which exceeds* 42° C."

In Drs. Mayer and Pilkington's article on "Some Problems in Pathogenesis in Schilder's Disease" in the November, 1936, number, on p. 822, para. 2, line 4, *for* "fatal after a few days", read "fatal after a few *months*".

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CONTENTS FOR JANUARY, 1937

PAGE

Original Articles—

The Seventeenth Maudsley Lecture: The Biology of Social Life; by *Sir Walter Langdon-Brown, M.A., M.D., D.Sc., F.R.C.P.* 1

Psychosis or Malingering? by *Frederick Dillon, M.D.Edin., and K. R. Masani, M.R.C.S.Eng., L.R.C.P.Lond., D.P.M.* 15

Therapeutic Narcosis with Soneryl; by *J. Stephen Horsley, M.R.C.S.* . 25

The Excretion of Sodium Barbitone in Patients Suffering from Mental Illness; by *E. H. Kitching, M.B., Ch.B.Leeds, M.R.C.S., L.R.C.P., D.P.M., and A. Dignam* 40

Some Observations on Endocrines in the Emotional Psychoses; by *J. L. Clegg, M.D., D.P.H., D.P.M.* 52

Irritability as a Symptom in Manic-Depressives; by *W. Mayer-Gross, M.D.* 61

Attempted Suicide: An Investigation; by *F. Hopkins, M.D.* 71

Reviews 95

A Preface to Nervous Disease; by *Stanley Cobb, M.D.*—Essentials of Psychopathology; by *G. H. Henry.*—Biological Time; by *Lecomte du Noüy.*—A Quantitative Analysis of the Structure of the Cerebral Cortex; by *S. T. Bok.*—Erbanlage und Epilepsie (Hereditary Disposition and Epilepsy); by *K. Conrad.*

Epitome of Current Literature 103

1. Anatomy and Physiology.—Psychology and Psychopathology.—3. Psychiatry.—4. Neurology.—5. Pharmacology and Treatment.—6. Pathology Biochemistry.

Errata 136

Also General Index to Vols. LXIX to LXXX, 1923 to 1934.

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THE
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No. 343 [NEW SERIES No. 307.] MARCH, 1937. VOL. LXXXIII

Part I.—Original Articles.

THE ELECTRO-ENCEPHALOGRAM IN EPILEPSY.

By F. GOLLA, F.R.C.P., S. GRAHAM, M.B., B.S., D.P.M., and W. GREY WALTER, M.A.Camb.

(From the Central Pathological Laboratory of the London County Hospitals for Nervous and Mental Disorders.)

IN 1929 Berger (1) discovered that changes of electrical potential in the human brain could be detected through the unopened skull. Since that time the study of electro-encephalography has occupied the attention of many workers, and the literature is already too extensive for adequate review in this place. A brief description of the technique for obtaining an electro-encephalogram, or "EEG", and a summary of its normal and pathological characters may be found in a communication by one of us (2) on the relation between the EEG and the presence of intracranial neoplasms. The cortex in the region of a tumour was found to produce abnormally slow potential waves, which were provisionally called "delta" (δ) waves to distinguish them from the normal "alpha" (α) waves which are the original "Berger rhythm". In the same paper a case was reported in which a focus of δ waves was found in the left parieto-occipital region associated with an area of degenerating cortex. The history in this case was of occasional minor attacks and one major fit and an indefinite severe illness in infancy, the only sign being a right homonymous hemianopia in accordance with the left-sided focus. Ether and nitrous oxide anæsthesia are also accompanied by the production of slow waves, but in this condition there is no fixed focus, the whole cortex being engaged in abnormal electrical activity. Since the publication of the above-mentioned results, a case of cerebral abscess has been examined, and the EEG was found to indicate a δ focus similar in character to those which have been found in cases of new growth.

These findings suggested strongly that the production of slow electrical oscillations is characteristic of cortex in some stages of dystrophy or degeneration, whether this condition be set up by new growth, inflammation or any other cause.

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In the course of the experiments at the Hospital for Epilepsy and Paralysis, Maida Vale, which resulted in the above observations, there was opportunity to examine 97 patients who complained of various sorts of epileptiform attack (Table I). In 39 of these patients the electro-encephalogram was found to be abnormal in so far as it exhibited a discharge similar to that associated with organic lesions of the cortex.* In 34 of these the abnormal discharge had a well-marked focus, while in 5 it consisted of a more generalized activity. In 25 cases the EEG could not be interpreted, either because a technical fault was suspected, or because the patient was so restless that no satisfactory records could be obtained. The majority of these restless patients were children less than five years old. Thirty-four patients gave EEGs which were within the normal range. The concept of the norm in electro-encephalography is discussed in the paper previously referred to ; briefly, we regard as normal an EEG which shows no regular waves of a potential greater than 5μV with the subject alert. The appearance of α waves in the parieto-occipital cortex when the eyes are open is not regarded as abnormal, although it is unusual, but the appearance of waves of this frequency (8–13 per second) anywhere but in the parieto-occipital region, or in this region when the subject is reading, is taken as an abnormal sign, since it has been found only in abnormal subjects. Actually, the only criteria which we have used to determine the abnormality of an EEG in this study of epileptics are : (*a*) The appearance of waves in any part of the cortex having a frequency of less than 6 per second and a potential with the routine leads of more than about 10μV, which are not affected by visual or mental activity ; (*b*) the appearance of waves resembling the α rhythm, but not originating in the parieto-occipital cortex and not affected by visual or mental activity. These criteria are, of course, empirical in that they are based on the fact that the characters concerned have not been found in any clinically normal subject under physiological conditions.† In the absence of an inductive explanation of the normal character of the EEG we must be content with the unsatisfactory state of the pathology of the subject. Nearly all the cases in this series were examined on several occasions. Several were examined every week for some months.

Although the number of cases included in this series is too small to provide material with statistical significance, examination of Table I yields some facts which are, at least, suggestive. Before discussing these, some explanation must be given of the terms used.

(1) " Positive heredity " indicates that the patient or a relative admits to the existence of an epileptic in the family. It is well known that comparatively few of those with a family history of epilepsy will admit it, even if they know of it, and many sufferers from epilepsy have not been told that

* This abnormal discharge should be clearly distinguished from that described by Gibbs, Lennox and Gibbs (9) as occurring during the actual course of a fit.

† Although Berger still considers that the α rhythm arises from the whole cortex, the balance of general opinion is strongly against this view.

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TABLE I.—*Electro-encephalograms of epileptic patients, series 2.*

	Number of cases.	Cases with abnormal EEG.								Cases with normal EEG.
		Total number.	Position of focus.						Diffuse.	
			Frontal.			Occipital.				
			Left.	Right.	Bilateral.	Left.	Right.	Bilateral.		
Total	72	38	9	6	8	4	3	4	4	33
Heredity positive	4	4	1	2	..	..	..	..	1	..
,, **negative**	68	35	8	4	8	4	3	4	4	34
Age: 1–9	7	6	1	..	1	1	..	2	1	1
10–19	21	13	2	4	2	2	1	2	..	8
20–29	22	10	4	2	2	..	..	..	2	12
30–39	17	9	2	..	3	1	2	..	1	8
40–49	3	1	..	..	..	..	..	..	1	2
50–59	2	..	..	..	..	..	..	..	..	2
Nature of fits:										
Major only	28	16	4	2	6	..	..	1	3	12
Major/minor	6	4	2	..	2	..	..	..	..	2
Minor/major	10	6	..	..	..	3	1	..	2	4
Minor only	23	11	2	4	..	1	1	3	..	12
Uncertain	5	2	1	..	..	..	1	..	..	3
Age at onset:										
Childhood	31	24	5	4	3	3	2	3	4	7
Adolescence	17	6	1	2	2	..	..	1	..	11
Later life	11	2	..	..	1	..	..	..	1	9
Uncertain	13	7	3	..	2	1	1	..	..	6
Clinical diagnosis:										
Idiopathic major	15	15	5	2	2	1	2	1	2	..
,, minor	8	5	..	3	1	1	..	..	..	3
Pyknolepsy	6	6	1	..	1	..	..	3	1	..
Narcolepsy	1	..	..	..	..	..	..	..	..	1
Congenital or early lesion	5	4	2	..	..	2	..	..	..	1
Traumatic	4	..	..	..	..	..	..	..	..	4
Syphilitic	1	..	..	..	..	..	..	..	..	1
Toxæmic	2	1	..	..	..	..	..	..	1	1
Functional	17	..	..	..	..	..	..	..	..	17
Doubtful	13	8	1	1	4	..	1	..	1	5

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others in the family have been afflicted with the same disease. For this reason the figures in this group should be suspected, though it may be significant that the four patients who gave a family history also gave abnormal EEGs.

(2) The majority of the cases were out-patients, so that the description of the fits was obtained from the patient or a relative. This fact undoubtedly admits of a certain amount of error.

(3) The age of onset, again, is derived from the patient or a relative. In many of the cases with onset in adolescence there was a history of suspicious convulsions as a baby, so that the number of cases with an onset in childhood is probably larger than is apparent.

(4) The clinical diagnosis is that applied by the out-patient physician or, in the case of the in-patients, the final diagnosis on discharge. Cases of cerebral tumour are not included in this series.

(5) The position of the focus in the cases with abnormal EEGs was determined with the triple amplifier cathode-ray oscillograph set-up as described in the paper mentioned above. The centre of the focus can be located to within about 1 cm., but this degree of accuracy was not attempted in all the cases, since it involves the taking of a considerable amount of film. It was considered adequate to establish in which part of which lobe the abnormality was to be found. The frontal foci were nearly all in the upper part of the frontal lobe, over the " area 6 a β " of Vogt. It is interesting that Foerster (3) attributes considerable importance to this area in the pathogenesis of epilepsy. Bilateral foci are those in which *symmetrical points* in the two hemispheres exhibit abnormal activity. The occipital foci were mostly in the regions of " area 19 " and " area 7 " of Vogt, except in the cases of pyknolepsy, when they were more posterior. The " diffuse abnormalities " were those with either multiple foci or with a general abnormal discharge over the whole cortex. A considerable degree of dementia was presented by two of these cases, of which one had a marked epileptic heredity ; the third was in *status epilepticus* ; the fourth was toxæmic, and the fifth was of doubtful diagnosis.

The fact which this table illustrates most clearly is that the majority of the cases with abnormal foci are those with an early onset of major fits to whom the diagnosis of idiopathic epilepsy had been applied, and who at the time of examination were less than forty years old. All 15 of the cases of major idiopathic epilepsy exhibited foci ; none of the 17 functional cases did so. On the other hand, all 6 of the pyknolepsies also gave foci, and the fits in these cases must certainly be classed as minor. There is a marked difference, however, between the records from pyknoleptics and those from a typical idiopathic epileptic. Fig. 1 shows the discharge from the frontal cortex of an epileptic girl, æt. 12. The focus is in the region of " area 6 a β " of Vogt. The discharge is of irregular frequency and amplitude. There was a less well-marked focus in the same area on the other side. This patient had chiefly major fits, as many as three a day without sedatives, and one a day or so while

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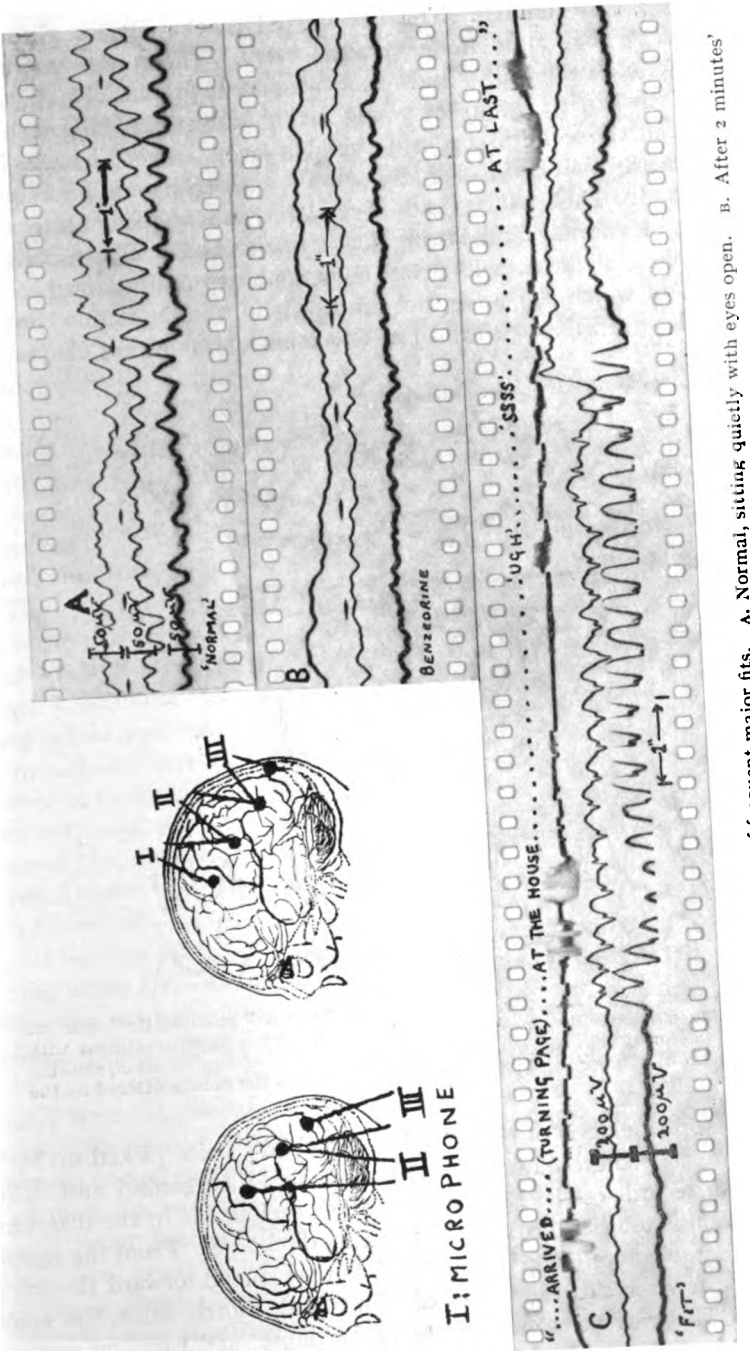

FIG. 1.—EEGs from a girl, æt. 12, with a history of frequent major fits. A. Normal, sitting quietly with eyes open. B. After 2 minutes' hyperpnœa.

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taking 2 to 3 gr. of luminal. The fits started at the age of three and had become more and more frequent and severe. There was an aura of spatial contraction. Clinically and electro-encephalographically this is a case representative of the group of young major idiopathics. Fig. 2A shows the discharge from the occipital cortex of a child of seven. This originates in the region of " area 18," and consists of large (50μV) smooth waves of a frequency of 3½ per second. This patient had very slight and frequent attacks consisting merely of vacancy and lasting for a few seconds. She usually had about ten of these slight attacks in an hour, and two or three rather more severe attacks in which she was quite unresponsive to stimulation, but still did not fall down. Fig. 2C shows the development of one of the slighter

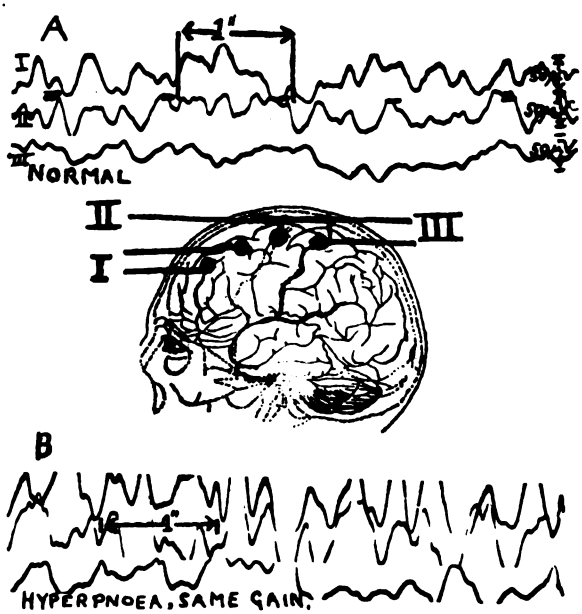

FIG. 2.—EEGs from a girl, æt. 7, with a history of frequent " pyknoleptic " fits. A. Normal, sitting quietly with eyes open. B. The same after 3 days' treatment with benzedrine. C. During the same examination as A, but with less amplification, showing the development of a minor fit. The top oscillograph shows the sounds uttered by the patient.

attacks. The top tracing is of the sound of the child's voice picked up by a microphone as she read aloud from a story-book. The second and third are oscillograph records of the EEG from the left occiput, as in the diagram. The amplification is too small to show the resting discharge. From the region of " area 18 " large waves (300μV) start, and as they spread forward the voice stops. After a few seconds the waves die down and, shortly after, the voice starts again. The patient was unaware of her lapse, and went on reading as if there had been none. In form these waves are similar to those from their

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area of origin in this patient between fits, but they are about four times larger. Both "resting" and "fit" discharges resemble the waves produced by ether anæsthesia in the normal subject, and this resemblance may be connected in some way with the fact that these attacks are completely resistant to sedation with luminal, suggesting rather that a cortical stimulant might be required in this condition. Acting on this, the effect of administering benzedrine sulphate 2 mgm. *b.d.* was tried. After three days of this treatment the fits stopped completely and the abnormal waves also vanished (Fig. 2, B). This experimental treatment and the significance of its results is discussed later, and will be dealt with in full in another paper.

The next point of interest is that none of the four cases with a history of trauma gave an abnormal EEG. One case, which was examined at the suggestion of Dr. Knight, of St. Bartholomew's Hospital, was supposed to have fits as the result of a blow in the right frontal region, but showed a focal abnormality in the right occipital cortex. Although this might have been the result of a *contre-coup* injury, it seemed an anomalous finding, so the early history of the case was gone into and was found to suggest that fits had, in fact, occurred before the accident.

The cases with a congenital or early organic lesion and abnormal EEG belong to a different group. One of these was the case previously reported, which showed a left parieto-occipital focus corresponding with a multilocular cavity communicating with the ventricle. Two cases had a history of early encephalitis, and one had a congenital nævus in the left frontal region. The history of the case with a normal EEG is obscure. The single case with a history of congenital syphilis had attacks which involved the right hand and arm only, and were preceded by a definite sensory aura in this region. In spite of the most careful and repeated examination of the area in the left parietal region suggested by the nature of these attacks, no abnormality could be discovered.

Of the two cases of toxæmic convulsions, one showed a generalized abnormality in the EEG similar to that found in cases of raised intra-cranial pressure. This case has been previously described (Walter (1937), Case 8). The other showed no abnormality and the diagnosis of toxæmia was somewhat uncertain.

Of the cases with doubtful diagnoses, the majority of those with abnormal EEGs were considered by their physicians to be probably idiopathic, while most of those with normal EEGs seemed to be of the functional type. These thirteen do not, however, contribute as yet to the attempt to correlate diagnosis and prognosis with the electro-encephalogram.

Although the conditions at the Hospital for Epilepsy and Paralysis, Maida Vale, were excellent for the continuous and repeated study of a few patients, it was realized that the examination of larger numbers of well-classified epileptics was required before these results could be seen in their correct proportion.

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The material offered by the London County Mental Hospitals was accordingly drawn upon, patients being conveyed to the Central Pathological Laboratory by omnibus.

TECHNIQUE.

Since at least 100 patients were to be examined, the apparatus used was designed to give a quick result at the expense of accuracy of location. Two amplifiers were used, both similar in design to those at Maida Vale, but the output stage in both was a low-frequency pentode driving writing oscillographs. These were made up from scrap Weston moving coil relays (Model 30). By fitting a stronger control spring and adding a light straw lever, these instruments were made suitable for writing on a large Palmer kymograph. Their undamped natural frequency was about 40 per second, and they were critically

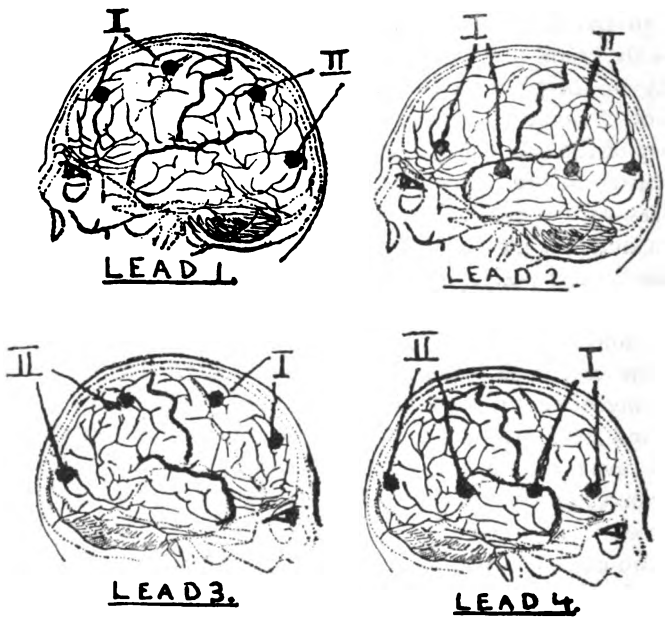

FIG. 3.—The routine electrode arrangements for the examination of patients in Series 2.

damped with oil. The overall time constant of the amplifiers was of the order of 1 second, so this arrangement was appropriate for recording electroencephalograms, the potentials of interest in this work having frequencies between 2 and 10 per second. The electrodes and the manner in which they were held on the patient's head were similar to those used in the first series of observations.

With only two recording systems the accuracy of location with the routine leads is to about 5 cm., and this was judged adequate for the purpose of these observations. It was at least possible to determine in which lobe and on which side a discharge was taking place. It is of interest to note that in one case (57 F.) in this series the accuracy of the double-writer system could be

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checked against that of the triple cathode-ray set-up, and was found to be completely satisfactory, the centre of the focus being correctly placed. The only difference was that with the triple machine it was possible to delimit the extent of the focus, which in this case was unusually small.

The time taken for the complete examination of a patient was from 15 to 20 minutes and a provisional interpretation of the records could be made at once. As a routine, at least four records were taken (Fig. 3), and usually the effect of opening and shutting the eyes was studied, particularly when a discharge was found in the post-central region, but not all the patients were able to co-operate in this respect.

Statistical Survey of Records.

One hundred and seventeen cases in all were examined on a single occasion, all of them residents in mental hospitals and all but two certified. They were unselected except in so far as behaviour went, only the reasonably docile and manageable type of case being sent up for examination. The medical officers of the respective institutions from which these cases were culled were requested to send typical cases of epilepsy, both major and minor, who would be likely to co-operate sufficiently well for records to be obtainable from them.

The records were studied independently by three observers, and were ultimately classed as positive, negative and doubtful, according to whether they did or did not show the type of curve which has come to be associated with epilepsy. The "doubtful" group consisted of records about which the observers were unable to come to an agreement or which, although not normal, did not show the characteristic features sufficiently clearly to be classed as positive. These records, ten in all, are not taken into account in the present survey.

Of the remaining 107 cases, 52 gave positive and 55 negative records; in other words, about 52% of insane epileptics selected at random give normal electro-encephalograms.

In order to discover any possible factors which would tend to make for positive and negative curves respectively, the case-histories of all patients were subjected to careful scrutiny, attention being paid, amongst other things, to hereditary factors and to the kind of treatment (notably sedatives) which the patients were receiving.

The results of this investigation can briefly be set out as follows:

(1) Minor epilepsy is far more frequently associated with negative records than major epilepsy. Only a small proportion of cases were exclusively one or the other, the majority showing a preponderance of either major or minor attacks. The exact numerical data will be found in the tables, but on the whole cases with exclusively or predominantly minor attacks belong to the negative group.

(2) The more a case tends to conform to the type known as idiopathic epilepsy, the more likely the record is to be positive. As criteria of idiopathic epilepsy were used: (*a*) Data from the family history so far as obtainable,

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notably incidence of epilepsy in parents and siblings ; (*b*) age at onset of attacks, infancy to puberty being regarded as the commonest age-group during which idiopathic epilepsy might manifest itself ; (*c*) absence of other causal factors and complications, such as trauma, cerebral tumours or operations and specific infections ; (*d*) some weight was given to congenital mental deficiency as indicating a tainted heredity. Cases with positive family histories and with no complicating conditions, in whom epilepsy manifested itself at or before puberty, provide the bulk of the typically positive records.

(3) Other things being equal, the greater the age of the patient at the time of examination, the more likely the record is to be negative. This only partly implies that the curves are negative, because the older the patient the more long-standing the disease. As a matter of fact, it is very difficult to assess the effect of duration of disease alone as a factor ; our group of middle-aged epileptics with negative curves includes a variety of cases, cases where there is an overlapping of factors as it were, so that it is difficult to make an unqualified statement as to influence either of age or of duration of disease separately. On the whole, however, as will be seen from the tables, the age-group 40 to 60 tends to show far fewer positive curves than the age-group 20 to 40. In trying to discover the possible reason for this, one might speculate along two different lines : firstly, is it that degenerative changes in the cortex incidental to age or to repetition of attacks over a period of years tend to obliterate signs of activity, both normal and abnormal ; secondly, is it that we are dealing with an accidental contributory factor—that, for example, a certain kind of epileptic tends to live longer than another, and that our middle-aged group therefore represents a picked population. There would seem to be some evidence in favour of this view ; as we have seen, more than one type of case tends to give negative records, notably cases with predominantly minor fits and cases in whom epilepsy first appeared in adult life. As major epilepsy does have a mortality, in *status*, one could quite fairly assume that cases who are less liable to develop *status epilepticus* are correspondingly more liable to survive to old age. In that case age as a factor in itself ceases to be of significance, and we are left with minor epilepsy and epilepsy due to other than hereditary causes as the two chief groups with normal electro-encephalograms.

(4) Administration of sedatives, principally bromides and luminal, does not appear to affect the electro-encephalogram in any way. With a very few exceptions all the patients were receiving bromide or luminal, some both, and the total dosage in the positive group is only a trifle higher than that in the negative group. This is, if anything, the reverse of what might be expected, considering the effect of these drugs on the frequency of fits ; but it is hardly significant, inasmuch as both the heavier dosage and the greater incidence of positive records are effects of a common cause, namely, a predominance of major attacks.

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TABLE II.

Clinical classification of all cases.	Total 52 positive records.		Total 55 negative records.	
(1) Nature of fits :				
(*a*) Major only	12	37	1 (æt. 62)	19
(*b*) Major chiefly	25		18	
(*c*) Major and minor	12		10	
(*d*) Minor chiefly	3	3	19	26
(*e*) Minor only	..		7	
(2) Age of onset of fits :				
(*a*) Infancy	13	38	9	26
(*b*) Childhood	25		17	
(*c*) Adult life	4		22	
(*d*) Unknown	10		7	
(3) Present age :				
14–18	1		1	
19–28	20	42	6	17
29–38	21		10	
39–48	4		18	
49–58	3	7	14	38
59–68	..		6	
Unknown	3		..	
(4) Presence and nature of aura :				
(*a*) None	3		25	
(*b*) Diffuse (dizziness, faintness)	5		15	
(*c*) Fright	1		1	
(*d*) Sensory—vague (choking)	6		3	
(*e*) Headache	1		5	
(*f*) Sensory—local	1	1	4	6
(*g*) Motor—local	..		2	
(5) Causation :				
Presumed idiopathic	42		13	
Due to focal lesions	2		23	
Cause unknown	8		19	

These tables were examined statistically, using the χ^2 method, and they show that in every case a definite relationship exists between the type of electro-encephalogram and the factor tested.*

The contentions made on page 145 are therefore substantiated, and it might be of interest to analyse the main group of exceptional cases of presumably true idiopathic epilepsy which yet gave negative records (Table III). We find that they represent instances of overlapping of significant factors, as has already been suggested, and it would appear that two such factors, namely, age of patient and nature of attacks, seem to dominate over the factor of causation of disease.

It will be seen that out of the total of 13 cases only 2 remain which show clinical features such as should place them in the positive group. One of them, a woman, is described by the medical officer in charge as a hysterical personality, and there is some doubt as to the true nature of the fits ; the other is one of the

* Thanks to the courtesy of Dr. Julian Blackburn we are able to quote the actual factors, which show that the odds against a chance relationship are as follows : Groups (1), (2), (3), (5) as 100,000, 4,000, 33,000, 1,000,000 to 1 respectively.

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TABLE III.—*Analysis of Cases of Presumed Idiopathic Epilepsy with Negative Records. Total Number of Cases*, 13.

Identification number.	Present age.	Nature of fits.	Age of onset of fits.	Remarks.
24/F.	53	..	..	..
43/F.	53	..	..	..
28/M.	50	..	..	..
9/F.	44	..	..	..
16/F.	42	..	..	..
41/M.	40	..	..	..
58/F.	39	..	..	..
13/M.	39	..	..	..
20/M.	39	..	..	..
48/M.	37	Minor only	..	..
51/F.	33	Minor +++	..	..
54/F.	27	Major and minor	Infancy	Hysteria
10/M.	23	Major ++	16	Focal aura

very few presumed idiopathic cases that show a local sensory aura—tingling in the feet. The rest of the cases would fall into the negative group on account of age and the nature of their fits.

Table IV is an analysis of the negative cases of Group 1, (*a*), (*b*) and (*c*)—

TABLE IV.—*Negative Cases with Major Fits. Total Number of Cases*, 29.

Identification number.	Details of fits.	Present age.	Age of onset of fits.	Remarks.
42/M.	Major +	65	..	..
26/M.	Major only	62	..	..
31/M.	Major +	58	..	..
23/M.	,, +	57	..	..
3/F.	Major and minor	57	..	..
25/M.	Major ++	53	..	..
28/M.	,, +	50	..	..
5/F.	,, +	50	..	..
21/M.	,, +++	49	..	..
16/M.	,, ++	47	..	..
20/F.	,, +	47	..	..
55/F.	,, ++	45	..	..
9/F.	,, +	44	..	..
11/F.	Major and minor	42	..	..
16/F.	,, ,,	42	..	..
35/F.	Major +	41	..	..
25/F.	Major and minor	41	..	..
41/M.	,, ,,	40	..	..
20/M.	Major +	39	..	..
40/M.	Major and minor	39	..	..
17/F.	,, ,,	37	23	Non-idiopathic
19/M.	,, ,,	37	15	..
52/F.	,, ,,	36	1	..
44/F.	Major ++	36	25	Non-idiopathic
38/F.	Major and minor	34	25	,,
54/F.	Major ++	27	Infancy	Hysterical
37/F.	,, +	26	13	..
10/M.	,, ++	23	? 16	..
2/M.	,, +	14	6	Meningitis

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—cases, that is, with major fits who should, on that factor alone, belong to the positive group. These also are presented in order of decreasing age, clinical data being given only in those under 39, who are therefore exceptions in a more strict sense of the word.

It will be seen that there are 5 cases which seem to be in every way exceptional, and two of them (54/F., 10/M.) are the same two which appeared as exceptions in the previous table. Two others again are reported as hystero-epilepsy in whom some of the fits are entirely hysterical. The remaining case (19/M.) had had a fit on the morning of the examination, so that it is just possible that the record was taken during the "silent period"—an interval following a major fit during which abnormal activity seems to subside so that very few or no potentials can be led off.

It is interesting to note that earlier authors (Gowers (10), Binswanger 13)) describe two groups of cases differentiated on the basis of high or low incidence of a local aura and focal features generally. The two groups correspond strikingly to our groups of negative and positive cases respectively, the former being those that are not instances of essential or idiopathic epilepsy and in whom minor fits predominate; the latter, having a clinical history which strongly suggests idiopathic epilepsy, and suffering predominantly from major attacks.

The group of positive cases gives records as follows:

Diffuse δ activity over the whole cortex . . .	6
Bilateral δ foci	26
δ focus on left	10
δ focus on right	10
Total	52

None of the cases with unilateral foci have a local aura or show any localizing features clinically; their case-histories differ very little from those that show bilateral foci and diffuse abnormality, and they all, with two known exceptions, belong to the class of idiopathic cases. One of the exceptions is a woman, aged 27, who has had fits for six years and who shows a well-defined focus in the left parietal region; the other is a case of exophthalmic goitre (woman, aged 37) who has had fits for the last two years only and who shows an extensive abnormal focus on the right side. These two cases seem to suggest that there might be an early or "acute" stage of the disease, during which the electro-encephalogram of the symptomatic case resembles that of the idiopathic one. It is noteworthy that both cases have relatively short histories of fits—actually the shortest in the entire series here described.

Discussion.

At the present stage no definite pronouncement is possible as to the significance of the δ-wave foci in epilepsy. These foci may merely represent

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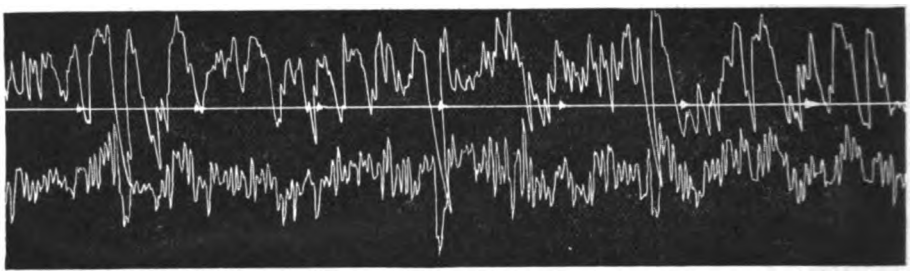

FIG. 4.—Case No. 57/F. Left parietal δ focus. Record taken from left (top line) and right (bottom line) parietal regions. Irregular 4-second δ waves shown on the left. From a symptomatic case, æt. 27, who has suffered from major fits for the past six years (one of the exceptional cases referred to below). Centre line. Time, 1 second.

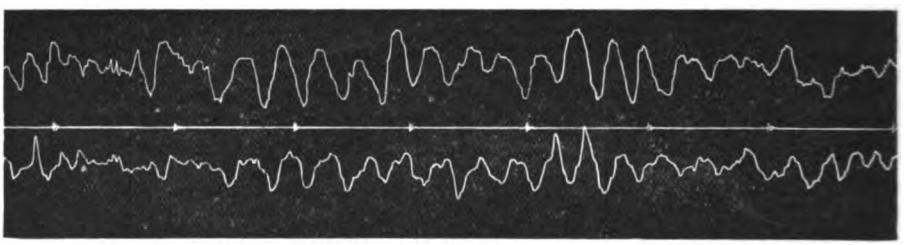

FIG. 5.—Case No. 10/F. Diffuse bilateral δ focus. Leads taken from left fronto-parietal (top line) and left parieto-occipital (bottom line) regions. Fairly regular 4-second δ waves shown in both leads. From a typical idiopathic case with predominantly major fits and no aura, æt. 21. Centre line. Time, 1 second.

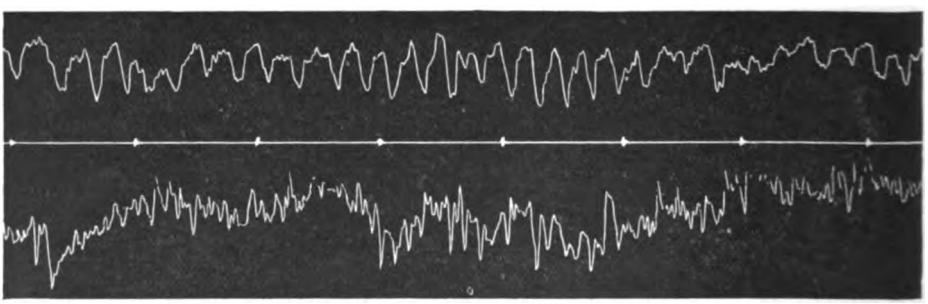

FIG. 6.—Case No. 46/M. Left frontal δ focus. Record taken from left fronto-parietal (top line) and left parieto-occipital (bottomline) leads. Top lead shows almost persistent 3–4-second δ waves. From an idiopathic case, æt. 34, with major fits only. Centre line. Time, 1 second.

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the electrical signs of degenerative processes in the cortex occurring as a result of epileptic fits, or they may indicate the existence of epileptogenic areas from which the fits are originated when appropriate conditions obtain.

There is no lack of evidence that the epileptic state may occasion extensive anatomical disturbance in the cerebral cortex, and some histological changes are found in about 50% of cases of epilepsy. It is unanimously agreed that the changes in the cornu ammonis and the cortex of the cerebellum described by all histo-pathologists who have investigated the pathology of epilepsy are attributable to the circulatory disturbances accompanying the fit. They possess no pathogenic significance other than that of being evidence of previous anoxæmia of the central nervous system.

The significance of the marginal gliosis first described by Chaslin, in 1889 (4), is apparently similar to that of the sclerosis of the cornu ammonis and the cerebellum. Both the marginal gliosis and the ischæmic generalized nerve-cell degeneration that precedes it and was described by Alzheimer (5) are found not only in essential epilepsy, but in cases of symptomatic epilepsy of some duration, in familial chorea, whooping-cough with convulsions and eclampsia gravidarum. The changes in the cerebral arterioles described by Weber (6) are interesting as affording some additional support to the view that these sclerotic changes are all of ischæmic origin and are sequelæ of the epileptic fit. The cortical sclerosis is always diffusely distributed—the whole of the cerebral hemispheres and the mid-brain may be involved, or random patches of sclerosis may be found over both hemispheres. In none of the cases that have been described is it unilateral or confined to a single area.

The fact that δ-wave foci were found almost exclusively in undoubted cases of essential epilepsy and not in the cases of symptomatic epilepsy is a strong argument against the view that they are related to these secondary sclerotic changes. In the group of epileptics investigated at Maida Vale four cases of symptomatic epilepsy occurred, and in those from the mental hospitals twenty-five cases were considered to be symptomatic. None of the four cases from Maida Vale and none, with two exceptions, from the mental hospitals exhibited the δ waves. So far as the evidence of histopathology goes, the sclerotic changes secondary to ischæmia should have been at least as frequent in these cases as in those of essential epilepsy.

The fact that in a number of cases the δ foci were well defined and single is again presumptive evidence against their association with the diffuse secondary ischæmic changes. The evidence on this point is, however, far from conclusive. The nerve-cell changes originally described by Alzheimer (5), and confirmed by a number of recent observers, would undoubtedly appear to precede the gliosis, and at some stage such early cellular changes may be confined to the epileptogenis zones. Some observations by Penfield (7) tend to show that at any rate in some cases the vascular disturbance at the

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beginning of a fit is confined to the " trigger " area from which the fit originates. It is not impossible that an early sign of the ischæmic lesion may be the production of δ waves by the damaged cells in cases of essential epilepsy, whereas in the case of symptomatic epilepsy such vascular disturbances do not occur.

The fact that δ-wave foci are found more frequently in early life and become rare after forty would fit in with this view, for Scholtz (8) shows very definitely that the ischæmic nerve-cell changes seen in the young epileptic brain are not seen after the age of thirty, when the secondary gliosis has become the leading pathological feature. Evidence from two angles of observation might be of capital importance in any attempt to estimate the pathological importance of the δ-wave foci. Firstly, evidence as to the association of the δ-wave area with the epileptic discharge, and secondly, as to the relation of the area to the prodromal symptoms and focal beginnings of fits.

It must be premised that only those cases showing either a single well-defined δ area or one with a less well-defined mirror image on the opposite hemisphere would be available for these considerations, and this limits the number of cases of evidential value very considerably. In the nature of things, too, only a small number of cases are likely to exhibit epileptic discharges during the electrical examination. Gibbs, Lennox and Gibbs (9) have recently published some observations in which such discharges were recorded in the electro-encephalogram. They do not, however, appear to have made any attempt to localize the area from which the discharges arose. On ten occasions it has been possible to observe the electrical concomitants of such discharges in patients with well-defined δ-wave foci. In two of these cases it has been possible to demonstrate that the discharge arose either from the δ-wave area or in its immediate vicinity. The first of these cases was that of a girl, aged 12, with a δ-wave focus situated in the left frontal area. The patient suffered from both major and minor fits, and during the course of the examination epileptiform discharges were noted which could only have arisen in the proximity of the δ-wave area or in the area itself. The second case is that of the girl of 7 years described on p. 142. In two adult cases the discharge again appeared to arise in the immediate vicinity of the δ focus. In two other cases it was only possible to affirm that the epileptiform discharge originated in the same hemisphere as the δ waves. Four cases of the type usually diagnosed as pyknolepsy showed frequent discharges which could be demonstrated to arise either from or in the immediate vicinity of well-defined δ areas.

The second case furnished additional evidence pointing to an epileptogenous significance in her δ-wave focus which, if confirmed in other cases, should be of decisive importance. Considerations alluded to above, and which will be dealt with in detail in another paper, led us to try the effect of administering a daily dose of benzedrine (β-phenylisopropylamine) sulphate to a number of epileptic patients, one of whom was the little girl in question. During a fortnight whilst receiving this drug she was entirely free from fits, which had

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previously occurred many times an hour, and the δ-wave focus disappeared. Cessation of the drug was followed, after a few days, by reappearance of the fits and of the δ-wave focus. This concomitant appearance and disappearance of both fits and focus was verified again by observations over a second and third period of drug treatment, with a control interval.

The attempt to correlate the focal δ-wave areas with prodromal symptoms or local beginning of fits met with no success. We were unfortunate in having few cases with well-defined aura and none with a Jacksonian type of discharge among the cases with δ-wave foci. Although an aura is by no means a rare occurrence in cases of essential epilepsy, it is relatively infrequent when such cases are compared with those of symptomatic epilepsy, and focal motor beginnings are exceedingly rare in the idiopathic form. Cases showing δ-wave foci belong exclusively to the idiopathic group, and hence it will only be from examination of far larger numbers that information as to the correlation of aura and δ focus is likely to prove of value.

It must further be borne in mind that the electro-encephalogram only gives information as to electrical events originating in the cortex in immediate contact with the skull. It is probable that any foci of δ waves occurring on the base or the mesial surface of the hemispheres will be missed with our present methods. Thus the cortical area explored in our electro-encephalograms does not by any means represent the whole potential source of δ-wave foci. If the aura of epigastric sensation, which is, according to Gowers (10), the commonest form of aura, be, as Penfield suggests, localized in the posterior inferior mesial portion of the frontal lobes near the ventral nuclei of the tuber, its presence could not be signalized by the occurrence of a δ-wave focus in the electro-encephalogram.

Any attempt to correlate the δ-wave foci with an epileptogenic or "trigger" area in cases of essential epilepsy may appear to be unwarranted when the existence of such "trigger" areas is not susceptible of direct proof. The hypothesis that such areas may exist in all cases is, we think, justified by the admitted occurrence of occasional cases of essential epilepsy with definite sensory or motor prodromata or of a definite local motor commencement of the fit. Experiments have abundantly shown that such definite prodromata are related to the existence of a "trigger" cortical zone, stimulation of which will evoke either the aura or the fit. Once such epileptogenic zones have been admitted to exist in the cases with definite prodromata, the fact that only a small minority of cases of essential epilepsy exhibit strictly localized prodromata is readily understandable. The area of the cortex from which localized responses can be elicited by stimulation is only a small portion of the whole. If localized δ-wave foci correspond to epileptogenic areas, their study will be of the greatest importance as affording the only means of discovery of such areas in the "silent" portions of the cortex.

Inasmuch as the aura or local beginning of a fit remains constant for very

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long periods, one would expect a similar constancy in the delimitation of the δ area if there be any relation between the two series. In the cases with well-defined δ-wave foci which were repeatedly examined for several weeks at Maida Vale the position of such foci has remained constant. In two cases of epileptic children and in all the cases of pyknolepsy the δ-wave foci never exhibited any other electrical discharge than that of the slow δ waves. All the other cases of similar foci only showed interrupted periods of δ-wave activity, varying from bursts of a few seconds' duration to an occasional group of three or four waves. This irregularity is very reminiscent of the spasmodic production of bursts of the *a* waves from the area 19 in normal subjects.

No conditions of cortical activity or stimulation such as influence *a*-wave activity have been found to have any influence on the occurrence of the δ waves. In the intervals between the bursts of δ waves the cortical discharge is not distinguishable from that of the surrounding normal area. Observations on a few cases have furnished evidence of occasional slight shifting of a δ-wave focus, such as has been shown by Adrian (11) to occur in the *a*-wave focus.

The frequency of these large slow δ waves varies in the course of a single examination; bursts of waves with a frequency of 2 per second may be succeeded by others with frequencies mounting up to 5 or 6 per second. In cases of pyknolepsy, however, the frequency of the δ waves remained constant. In these cases the commencement of an epileptogenic discharge was heralded by the slowing of the constant δ-wave frequency. The slowing was generally from about 4 to 3 beats per second. The amplitude of the waves is increased by over-breathing, but in a single series of experiments was not found to be affected by amyl nitrite inhalation or by carbon dioxide in the inspired air.

Further experiments on the effect of drugs will be dealt with in a subsequent publication. At this juncture we only wish to note that there was no evidence that the administration or withholding of bromides or barbiturates in the doses used in attempting to control epilepsy had any influence on the electro-encephalogram.

The localization and number of δ-wave foci is illustrated in the accompanying tables. Single foci or single foci with a "mirror" focus on the opposite hemisphere are noted most frequently in the young children and early cases in Maida Vale Hospital. The occurrence of a "mirror" focus in a number of cases is consonant with the observations of Gozzano (12) on rabbits, in which a localized epileptic discharge produced by application of strychnine to the cortex evoked a "mirror" discharge in the opposite hemisphere which could be abolished by section of the corpus callosum. In many of the cases, however, and particularly among the essential epileptics from mental hospitals, multiple foci are noted, although there is no apparent relation between the occurrence of multiple foci and the degree of dementia. In some of the cases the impression was given that the whole cortex was liable to display δ-wave discharges at intervals.

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It is an old observation that in long-standing cases of epilepsy there is a tendency for strictly localized prodromal symptoms to be lost, and for fits to occur either without prodromal symptoms or with such symptoms varying from one fit to another, suggesting a multiplication and scattering of the hyperexcitable cortical areas. The infrequent occurrence of δ-wave foci in cases of essential epilepsy after forty is illustrated in both the Maida Vale and the mental hospitals cases.

SUMMARY.

(1) Electro-encephalograms have been taken from two series of epileptic patients, comprising 214 cases altogether. In 91 of these the EEG was considered to be abnormal, in most cases on account of a discharge of slow δ waves.

(2) In both series a correlation was found between the type of EEG and the clinical diagnosis, idiopathic epileptics with a history of major fits providing most of the abnormal EEGs.

(3) A correlation appears to exist also between the EEG and the age of the patient, few abnormal EEGs being found in patients over the age of forty.

(4) In most of the cases in the first series the abnormality was found to be persistent and localized.

(5) EEGs have also been taken during the onset and course of fits. In some cases the discharge characteristic of the fit was found to start in the same region as the persistent abnormality.

(6) The significance of these findings is discussed.

We wish to record our thanks to the medical superintendents and medical officers of the London County Mental Hospitals, and to the medical staff of the Hospital for Epilepsy and Paralysis, Maida Vale, for their kind help in supplying us with clinical material, and for putting such information as we required at our disposal.

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THE DIFFERENTIATION OF NEUROSES AND MANIC-DEPRESSIVE PSYCHOSES.

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> " We are now but just awakening from the metaphysical delusion that diseases are separate entities ; and we have scarcely rubbed our eyes free from the tendency to see in each disease, or even in each stage of a morbid process, a fixed species, having no genetic affinities to any other."
> CLIFFORD ALLBUTT, *St. George's Hospital Reports*, 1866, i, p. 61.

INTRODUCTORY : THE PROBLEM.

THE title of this paper will suggest a number of problems which are still the subject of considerable controversy.

Thus, some authorities appear to uphold the general proposition that there is a fundamental and clear-cut distinction between all neuroses (or psychoneuroses) on the one hand, and all psychoses on the other.

Other authorities, whilst not disputing that it may be useful and convenient to make a broad distinction between these two categories, deny that it is always possible, or profitable, to make the sharp distinction which is thus advocated.

Should it transpire that this latter view is the correct one for our special field of the differentiation of the neuroses and the manic-depressive psychoses, the general proposition of a fundamental distinction between all neuroses on the one hand, and all psychoses on the other, would be difficult to maintain ; and any general deductions that might be drawn would become suspect, or need modification.

It might, of course, still be held that there were clear-cut distinctions in other fields between what seemed to be closely allied groups.

Thus, for example, it might be held that " schizoid psychopathy " is, so to speak, contiguous to definite schizophrenia, but that it is not continuous with it. But it is evident that if no fundamental distinction between neuroses and manic-depressive psychoses is found to exist, the validity of a general neurotic-psychotic dichotomy of a sharply cut kind would be impaired.

Now our special problem of the differentiation between neuroses and the manic-depressive psychoses is probably the favourite field in which this dichotomy is made.

British psychiatrists in particular have shown much interest in this special

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problem, and attention has been focused upon it by public discussions and popular expositions, as well as in articles with a more limited appeal. There is certainly complete agreement that there are many cases where the problem does arise.

Unfortunately, the same agreement is not shown as to the terminology that should be employed to describe these cases, and there is a consequent uncertainty as to exactly what types of patients are being discussed.

Thus, some authors speak of "neuroses" or "psychoneuroses" as a group without further differentiation; the majority separate out from the great band of the "neuroses" syndromes which they term "psychoneurotic", "neurasthenic" or even "reactive" depressions; one leading authority speaks only of "neurasthenia"; and hardly any explicitly mention "anxiety states" in this connection.

It is evident that it might be argued that a number of separable questions are, in fact, raised which call for differentiation and separate treatment.

Thus it might be maintained that the "neuroses" of the title, or the "neurasthenia" of the upholder of this term, should be subdivided, and that the differentiation from the manic-depressive psychoses of the syndromes thus constituted should be considered one by one.

The differentiation of depressive states belonging to what has been called "psychoneurotic" depression from depressive states belonging to the manic-depressive psychoses might be held to constitute one problem, and the differentiation of anxiety states from the manic-depressive psychoses might be held to constitute a different problem; indeed, judging from the lack of attention paid to it, this last differentiation might not be held to constitute a problem at all.

A subdivision of "neuroses" into different groups would clearly be a necessary preliminary if, in fact, it was maintained that neurotic syndromes could themselves be accurately and precisely defined and clearly differentiated from one another.

But with one possible exception, all British psychiatrists appear to agree that the neuroses cannot be sharply differentiated, and all emphasize the existence of intermediate or mixed forms, whatever classification of the neuroses they themselves may favour.

It is true that discussion has mainly circled round the differentiation of manic-depressive depressive states from "psychoneurotic" depression, or whatever term of a comparable kind is preferred; it is also true that such categories appear to be created by certain authors for the purposes of these discussions, and are not mentioned in their classifications of the neuroses given elsewhere.

But from what has been said it is evident that, for example, "anxiety states" are not considered to constitute a sharply separated group, and therefore cannot represent a sharply separated diagnostic problem.

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The omission of these cases as a differential difficulty is hard to understand, would be difficult to justify, and will not be emulated.

It is not, of course, maintained that the problem of differentiation from manic-depressive psychoses arises in connection with every neurotic case that is seen; but it is a problem which does arise with many of them. Bearing in mind, therefore, the general disagreement as to terminology, together with the general agreement that clear lines of demarcation between different types of neuroses are not to be found, it seems quite justifiable to speak of "neuroses" without further differentiation, as has been done.

The material for this investigation will readily be forthcoming by grouping together cases presenting the varied manifestations of anxiety and depression as the outstanding features, as it is with such cases that our problem does arise.

It is proposed—

(1) To give a brief review of the very divergent opinions which have been expressed as to how such cases can or should be differentiated, together with the reasons which some have put forward as to why a sharp division is for them of crucial importance.

(2) To select certain of the criteria which have been laid down as achieving this sharply cut differentiation.

(3) To make an attempt to apply these criteria to a group of cases which have been personally observed.

(4) To discuss the findings and to see what deductions can be drawn.

Views on the Possibility of a Sharp Distinction.

There can be no doubt that the question "Is this case neurotic or psychotic?" is widely considered to be a proper question as applied to cases lying within our field; and a proper question, not in the sense that it may have meaning, but in the sense that it must have meaning, as when two mutually exclusive alternatives are proposed.

This is certainly a view which is completely endorsed by many leading authorities in this country.

Thus Ross (1) divides the neuroses, for convenience, into three categories of neurasthenia, hysterical reaction and obsessional illness, emphasizing, as he does so, that much overlapping between his different groups is shown. His "neurasthenia" certainly includes what others would class as "anxiety states" or "psychoneurotic depressions", and it is by no means confined to the description of states of fatigue. He writes: "There is no 'borderline' between the 'depressive' (i.e., psychotic) and the neurasthenic, though there may be difficulties in diagnosis." Elsewhere, in a well-attended discussion on this very topic (2), he is reported as having informed the only speaker who disputed this view that, if that speaker would carefully examine

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mental states, this would "lead him to perceive fundamental differences between psychoses and neuroses".

Similarly Buzzard, in another discussion on our subject (3), said that "the distinction between the two conditions, although sometimes difficult, was of fundamental importance as regards prognosis and treatment, as well as in the interests of academic accuracy".

Yellowlees (4) and Gillespie (5) appear to be very definitely of the same opinion, and, in one of the discussions referred to, the latter stated (6) that "the view that there was a 'continuous graded series' between the neuroses (or rather the psychoneuroses) and the manic-depressive psychoses was not generally held".

This statement gives a misleading impression, for it excludes the many distinguished psychiatrists who are of an entirely contrary opinion.

Thus Mapother (7) writes: ". . . there is a complete gradation" (from the manic-depressive psychoses) "to certain minor disturbances which, whether continuous or phasic, are usually termed neuroses." Again Lewis (8), commenting on the view that there is a fundamental distinction, writes: "There can be little doubt that this view is false." He quotes Bumke and many other authorities in his support, and it is refreshing to note that, unlike several of the authors mentioned, he provides detailed factual evidence in favour of his contention. Again, Adolf Meyer (9), who has exercised such a profound influence on the younger generation of psychiatrists in this country, also most emphatically refuses to draw any sharp line, and in this is, of course, entirely representative of American psychiatric thought (10).

The Reasons Given for the Importance of this Distinction.

The reasons for the vital importance of the distinction, the possibility of which is so much disputed, have been put forward by Ross (1) with his customary lucidity. As regards prognosis he writes: "The depressive is quite sure to become well"—but will probably relapse—whilst "the neurasthenic must be made well; he does not tend to drift into good health as the depressive will. If properly cured, he should not relapse".

The treatment again is "entirely different". The neurotic—called neurasthenic by Ross—needs active psychotherapy, and does not get well without it. Attempts at active psychotherapy are, however, considered to be quite useless, when not actively harmful, for the "manic-depressive", who will become well in time if protected from suicide, whilst "the chances of the neurasthenic doing this are nil".

Similarly Yellowlees (11) is thus reported: "Melancholia was a psychosis, and it was *ipso facto* not amenable to psychotherapy. . . . He was referring to the word in its modern sense."

Again, Crichton-Miller (12), who prefers the term "cyclothymia", because

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" manic-depressive " smacks too much of the mental hospital, an institution with which he, no doubt correctly, repudiates ever having had any connection, stated: " True cyclothymia varies for endogenous reasons, and all other explanations are fallacies of lay observation." (It can, presumably, be inferred that " false " cyclothymia would imply those cases which are falsely supposed to be true—the fallacies of medical observation.) Crichton-Miller does not resist the temptation to speculate as to possible organic causes for " true cyclothymia ", and appears to favour some " thalamic " origin. He is not dogmatic as to what exactly this means.

Summary of Views.

It will be clear that contradictory opinions have been expressed with equal conviction. Some British authors hold that these cases can, and should, be divided into two sharply contrasted categories, two " fixed species ", the neurotic and psychotic. In the production of the psychotic, endogenous-physical-constitutional factors play a predominant, psychological factors a minor part. These cases, if protected from suicide, get well in the course of time for endogenous reasons, and, according to one author, " all other explanations are fallacies of lay observation ". They must be black-listed psychotherapeutically (" using the word in its modern sense "), for this is at best useless, and may be actively harmful.

In striking contrast stand the neuroses. Psychological factors are here predominant; constitutional and endogenous factors far less important. They " must be made well " by active psychotherapy and do not " drift into good health ".

Other authors maintain that there is a " continuous graded series ". They believe that there is, so to speak, a large band of grey between the black and the white, or, if you will, that many cases are piebald.

Their views on prognosis and treatment suffer from a corresponding lack of sharp definition, and could not be put forward in any attractively simple and schematic form. Any such formula as " Analyse a neurosis, guard a psychosis; don't guard a neurosis, don't analyse a psychosis; I cure the neurotic, time cures the psychotic ", would seem an undue simplification of the facts to be observed, and of the deductions to be drawn from them.

It is, nevertheless, to be feared that such a formula is scarcely a parody of views which are widely held, and a scarcely illegitimate inference from statements which leading authorities have made.

The Selection of Criteria.

It is proposed to select certain of the criteria which have been laid down as of value in the differentiation between neuroses and manic-depressive psychoses, and subsequently to make an attempt to apply these selected criteria to a group of cases which have been personally observed.

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In principle, this procedure would seem to be wholly legitimate ; for if it is claimed that a sharp distinction can be demonstrated by the application of certain criteria, it is hard to see what objection can be made if this claim is taken literally, and the criteria are, in fact, applied to see whether what is claimed is in fact achieved.

But in practice there are grave difficulties to be overcome in attempting to pursue such a course. One of these difficulties is certainly not that of finding criteria, for a comparatively brief search was rewarded by the discovery of some thirty of them, and no doubt a more careful search would have been rewarded by the disclosure of several more. The problem of selection from this mass is, however, a very different and a much more difficult matter ; and selection there must be, for, unfortunately, some of these criteria have been enlisted by different authors on different sides ; some of them do not appear to present a meaning which is clear and unambiguous ; some of them do not present any very obvious meaning at all ; and many, even when meaning can be discerned, are quite extraordinarily difficult to apply, if the assignment of a given case to either one or other of two sharply contrasted categories is the avowed aim, as it is.

Thus, for example, contradictory opinions have been expressed as to the implications to be drawn from the fact of, or reaction to, good sleep. Again, loss of the capacity for " reality testing " does not convey that precise meaning to those who are not psycho-analysts that it may be presumed to have for those who are accepted as such. Moreover, this faculty of " reality testing ", apart from any difficulty in understanding what it means, is evidently a possession or attribute which is shown in varying amounts. It is clearly a grave objection against the value of a given criterion if that criterion is of such a type that, when it is applied to a particular case, no categorical result can necessarily be anticipated, when the object of its application is to achieve such a result.

This same objection can be levelled against many of the proposed criteria, and is very well illustrated by " insight ", which both for this reason, and because of its popularity, merits further attention. It is true that Yellowlees, at the discussion on our subject held by the Psychiatric Section of the Royal Society of Medicine (11), referred to the " final and subtle touchstone, namely the presence of insight in the neuroses and its absence in the psychoses. . . . He had never known that touchstone to fail ". But this statement would seem to indicate the possession of a private insight which is far from being public property, for, as Lewis has pointed out in his recent paper (13), " insight " is not a simple attribute which is either present or absent. On the contrary, insight is a very complicated conception, and to have insight into an illness involves, amongst other things, not only a recognition of the fact of being ill, but an assessment of its nature. What degree of correct assessment and understanding constitutes insight ? Surely this is something which patients,

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and physicians, possess in most varying degree, and "complete insight" is necessarily a prerogative of the Almighty alone. Moreover, even in the vague, loose, but not entirely meaningless way in which the term is commonly employed, the claim made by Yellowlees seems demonstrably untrue, for insight can surely be strikingly present in certain "manic-depressive" depressions, whilst it is notorious that many neurotics are convinced that their symptoms are due to occult physical conditions which cannot be found.

For these reasons it was decided to exclude "insight" from the band of selected criteria. It cannot, however, be claimed that the same type of objection cannot be levelled against several of the criteria which have been chosen, but this seemed unavoidable, as there were few criteria against which such objections could not be advanced.

Finally, it might be maintained by particular protagonists of the proposition that there is this sharp distinction between neuroses and manic-depressive psychoses to be demonstrated by the application of criteria (rather than by a process of divination)—that some or most of the selected criteria are not those by which great personal store was laid.

It is impossible to answer individual objections of this kind in a manner which will give satisfaction. All that can be attempted is to compile as

Table of Differential Diagnostic Points (modified from that of Gillespie (14)).

Psychotic.	*Neurotic.*
1. Presenting subjective complaint of depression.	1. Presenting subjective complaint of "anxiety" or "fatigue".
2. Somatic complaints absent or unimportant.	2. Somatic complaints prominent.
3. Remorse and self-reproach present.	3. Absent or "insincere".
4. Does not blame others.	4. Blames others.
5. Loss of weight "invariable".	5. Not invariable.
6. Constipation "invariable".	6. Not invariable.
7. Depersonalization present.	7. Absent.
8. "Adequate" precipitating factor often absent. "Cause" often not discoverable.	8. "Adequate" precipitating factor often present. "Cause discoverable or clear."
9. Steady course "strikingly independent of environment".	9. Unsteady variable course, dependent on environmental factors.
10. Healthy except for circumscribed attacks.	10. "Seldom absolutely well."
11. Positive family history.	11. Negative family history.

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representative a list of criteria as possible; for otherwise it would have been necessary to embark upon the examination of the results obtained from numerous tables, all slightly differing. But if it be alleged that the list which has been selected is unfair, and that the application of different criteria would have produced different results, this could readily be put to the test. It has not been possible to discover an instance where this has actually been done.

The Application of the Selected Criteria to a Group of Cases.

(*a*) *The material used.*—The patients were all personally observed at the Hospital for Epilepsy and Paralysis, Maida Vale.

Twenty-five consecutive patients were taken, who displayed the manifestations of anxiety or depression as their outstanding features. In none of these cases, so far as could be determined, was the picture significantly complicated by "organic" mental symptoms dependent upon arteriosclerosis or other obvious physical factors; and a similar refusal was made to admit cases complicated by epilepsy, or gross mental defect, or where schizophrenic features were to be found.

The material was thus carefully selected; and as the proportion of cases with a fairly severe degree of emotional disturbance was quite high—very much higher than in the psychiatric out-patient department of a general hospital like St. George's—it seemed rather suitable for the present purpose. Indeed, about one-third of the patients were specially referred for an opinion on the very point under discussion by either neurologists or those members of the staff exclusively engaged in intensive psychotherapy. Only two or perhaps three of these patients were "certifiable".

(*b*) *The application of the selected criteria to this material.*—The most cursory perusal of the table of the eleven criteria which have finally been chosen will disclose that these criteria are of different types, possess different values, that the exact meaning of some is ambiguous and the possibility of application doubtful.

It must, however, be realized that they have all been advocated by others, and that this is not the writer's fault. As doubt was experienced as to exactly what some of them meant, and as little help was forthcoming from those who advocated their use, it is necessary to make an attempt to explain what has been understood by them here.

(1) and (2). The first two criteria were taken to refer to the nature of the presenting complaint, or complaints, which were made spontaneously. Both the patients, and the friends or relations who accompanied them, were first of all invariably asked questions with the idea of eliciting why they had sought medical advice. A considerable discrepancy could be observed between what the patients themselves complained of primarily, and what most struck

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their friends and relations as being the matter with them. Thus the latter often started with the statement that the patients appeared "low spirited" or "depressed"; whereas the patient complained of other things—inability to settle or concentrate, or of various somatic discomforts and aches and pains.

It was presumed that, when there was a discrepancy, more attention should be paid to the statements of the patients than to the observations of their friends or relations.

If the primary complaint was of depression or "low spirits", then the patient was assigned to the "psychotic group"; but if the complaint was not so much of depression or low spirits as of tension and anxiety, or of what appeared to be expressions of these ("feeling on edge", "like screaming", etc.), then the patient was classed—for this purpose—as being neurotic.

When the complaint did not refer so much to emotional experiences, recognized as such, as to what were felt to be somatic experiences or discomforts, then the complaint qualified for the second diagnostic point.

Complaints of low spirits were not infrequently combined in the same sentence with somatic complaints. It will also be apparent that the assessment of what the patients said was a very subjective and often an extremely difficult matter.

(3) and (4). The presence or absence of self-reproach was repeatedly mentioned as a most valuable, and even on occasion a specific finding, pointing definitely in one direction or the other; but again exactly where shouldering blame, or its attribution elsewhere, becomes sufficiently marked to merit inclusion as a definite symptom is a nice issue which does not lend itself readily to clear rules. Very great variation as regards accepting blame or attributing this elsewhere was shown, and the two attitudes were not infrequently combined. In dealing with these patients, perhaps illegitimately, more definitely paranoid statements and attitudes were included under "blames others" as well as those more vague projections, or attempted refusals to accept responsibility, which probably alone were meant by those who laid down this criterion. But again it seemed impossible to draw a sharp line.

(7). It seems unquestionable that there is every possible gradation between what is sometimes called "affective loss", i.e., feeling hard-hearted, etc., and "depersonalization" or "derealization" in the more strict unreality sense. All these varied manifestations were included under this heading.

(8), (9) and (10). With all these three criteria the same difficulty arises. What is an adequate precipitating factor or cause? What amount of reaction to environmental factors must be shown to qualify as "neurotic reactivity"? By what standard must the past health be judged? Definite guides on these points are lacking, but an attempt was made to take a "common-sense" view. No doubt, in many instances, others would have formed different decisions.

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Scoring.—Considerable generosity has been displayed in awarding positive and negative marks. The times when doubt occurred were numerous; but it was felt better to try to see things as much as possible in terms of black and white, rather than in what seemed to be more accurate but intermediate shades. Thus, "some reaction to environmental factors" was evident in every case; but several cases were marked as if this was not so if the reaction appeared to be small. The method of scoring has been "Psychotic" +1, "Neurotic" −1, Uncertain or Contradictory 0. In the family history, only very definite breakdowns necessitating hospital care have been included, and it has not been possible to obtain a negative mark. Exception might well be taken to the marking of criteria 5 and 6, on the grounds that what is stated not invariably to be present should not be marked in a definite way.

	Case 1. ♀. M. 38.	Case 2. ♂. M. 44.	Case 3. ♀. M. 45.	Case 4. ♂. M. 34.	Case 5. ♀. S. 19.
1. Complaint of depression	0	0	0	0	+1
2. Other complaints, especially somatic	0	0	−1	−1	−1
3. Remorse	+1	−1	−1	−1	+1
4. Blame others	−1	+1	0	−1	+1
5. Loss of weight	+1	+1	−1	+1	−1
6. Constipation	+1	+1	−1	+1	−1
7. Depersonalization	0	−1	−1	−1	+1
8. Precipitating factor	−1	+1	+1	−1	+1
9. Variability	0	−1	+1	0	+1
10. Past history	+1	+1	+1	−1	+1
11. Family history	0	0	0	0	0

	Case 6. ♀. M. 55.	Case 7. ♂. M. 42.	Case 8. ♂. M. 53.	Case 9. ♀. M. 48.	Case 10. ♂. M. 38.
1. Complaint of depression	0	0	0	0	+1
2. Other complaints, especially somatic	−1	−1	−1	−1	+1
3. Remorse	0	−1	0	+1	0
4. Blame others	+1	−1	+1	−1	0
5. Loss of weight	+1	+1	+1	0	0
6. Constipation	−1	+1	+1	0	0
7. Depersonalization	−1	−1	+1	−1	−1
8. Precipitating factor	−1	−1	−1	−1	−1
9. Variability	0	+1	0	+1	+1
10. Past history	−1	+1	+1	+1	+1
11. Family history	0	0	0	0	0

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	Case 11. ♂. M. 24.	Case 12. ♂. S. 53.	Case 13. ♀. M. 63.	Case 14. ♀. M. 60.	Case 15. ♂. S. 40.
1. Complaint of depression	−1	−1	−1	−1	−1
2. Other complaints, especially somatic	−1	−1	−1	−1	−1
3. Remorse	−1	−1	−1	−1	+1
4. Blame others	−1	0	−1	−1	+1
5. Loss of weight	−1	−1	+1	+1	+1
6. Constipation	−1	−1	+1	+1	+1
7. Depersonalization	−1	−1	−1	−1	−1
8. Precipitating factor	0	+1	+1	−1	−1
9. Variability	+1	+1	+1	−1	−1
10. Past history	+1	−1	0	+1	0
11. Family history	0	0	+1	+1	0

	Case 16. ♀. S. 37.	Case 17. ♂. M. 41.	Case 18. ♀. S. 19.	Case 19. ♀. M. 26.	Case 20. ♀. S. 24.
1. Complaint of depression	−1	−1	+1	−1	−1
2. Other complaints, especially somatic	−1	−1	+1	−1	−1
3. Remorse	−1	−1	−1	−1	−1
4. Blame others	−1	+1	+1	−1	+1
5. Loss of weight	+1	+1	−1	−1	+1
6. Constipation	+1	−1	−1	−1	−1
7. Depersonalization	−1	−1	+1	−1	+1
8. Precipitating factor	−1	0	+1	+1	+1
9. Variability	−1	+1	+1	−1	+1
10. Past history	+1	+1	+1	−1	+1
11. Family history	0	0	+1	−1	+1

	Case 21. ♂. M. 56.	Case 22. ♂. M. 36.	Case 23. ♀. S. 27.	Case 24. ♀. M. 45	Case 25. ♀. M. 25.
1. Complaint of depression	−1	−1	+1	+1	+1
2. Other complaints, especially somatic	−1	−1	+1	−1	−1
3. Remorse	−1	+1	−1	−1	−1
4. Blame others	−1	−1	+1	−1	+1
5. Loss of weight	+1	+1	−1	+1	+1
6. Constipation	+1	0	−1	+1	+1
7. Depersonalization	+1	+1	−1	−1	+1
8. Precipitating factor	+1	−1	+1	−1	−1
9. Variability	−1	−1	+1	+1	+1
10. Past history	−1	+1	+1	+1	+1
11. Family history	0	0	0	0	0

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Discussion of the Findings and Deductions.

To work out an addition sum for each case would certainly be a piece of mathematical nonsense; but without the aid of spurious precision of this kind, a perusal of these tables does not disclose that the application of these criteria in fact achieves a sharp division into two groups, or anything like it.

It would, of course, be impossible for such a small series to show every conceivable combination of psychotic and neurotic "points"; but even this small series makes a very fair showing in this respect.

In other words, the application of these criteria does not appear to work. Only one deduction seems possible—that there is no sharp division as has been alleged, or if there is, that it cannot be achieved by these means. Indeed a sharp division in nature is scarcely to be expected, and, as regards the field under survey, it is not to be found by the means employed here.

What, then, are the positive deductions that can be drawn?

"*Disease entities*" *or* "*reaction types*"? Writing in 1866, the late Sir Clifford Allbutt stigmatized the belief that diseases were separate entities as a "metaphysical delusion", and his contemporary, Henry Maudsley (15), wrote of the psychiatric classifications in his day: "As it is not Nature which makes divisions, but man who imposes his conditions on it, the prudent might well not make too much of them".

The standpoint advocated by Allbutt and Maudsley so many years ago has more recently been expressed in the happy aphorism that "classifications are inventions and not discoveries", or, less epigrammatically, by the statement that the conception of "reaction types" has displaced that of "disease entities".

"Manic-depressive psychoses" and "neuroses" are, according to this view, held to be names given to syndromes; and the typical or "pure" case is only a case to which certainly many cases do approximate, just as in other spheres an approximation can be made to an ideal, but it is also a case to which a large number do not approximate.

These fundamental psychiatric platitudes were thus recently expressed (8): "The current conception of the purposes of psychiatric classification . . . essays to delimit only types of reaction with every possibility of intermingling and no certain independence. . . . 'Pure' syndromes or reaction types are less and less often found as more careful examination of the patient and his illness is made."

"*Disease entities*" *not found.*—Now exception has already been taken to the criteria which have been laid down as achieving the "fundamental difference" between neuroses and the manic-depressive psychoses on the grounds that, not only are they difficult to apply, but also that, when applied, no definite result is obtained.

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The danger of narrow formalism.—But it is evident that criteria which might help to differentiate, must also depict and delineate; and exception may also be taken to these criteria on this score.

The pictures of manic-depressive psychoses or of neuroses which are thus derived are far too dogmatic and formal, and represent a misleading simplification of the facts to be observed.

Taking, for example, the manic-depressive psychoses, Lewis (8), in his admirable and detailed study of definite cases, points out that the presenting subjective complaint in "melancholics" is frequently not that of depression. He further expresses surprise that anxiety, which he regards as "a common and probably integral part of the depressive reaction", is barely mentioned in the descriptions to be found in the current British text-books, such as that of Henderson and Gillespie (16), especially in view of the fact that the frequency and importance of anxiety in depressive states has been stressed by Bumke, Delmas, Lange, Mapother and others. Again, the majority of his patients made somatic complaints of so-called "neurotic" type—a point which is also stressed by Hohman (17) amongst others, who draws attention to the diagnostic difficulties involved when these complaints are in evidence, and the subjective complaint of depression is not.

Again, the majority of the 61 cases studied by Lewis did not shoulder full responsibility, but projected or allocated it elsewhere. Many did not show self-reproach; self-reproach and paranoid attitudes were frequently combined, or alternated rapidly. Lewis also strongly dissents from the view that these "psychoses" necessarily run a course independent of environmental factors; in only nine of his cases could this be held to be so. In his recent study Mayer-Gross (18) has pointed out that depersonalization is not a prerogative of any special type of reaction, and that it unquestionably occurs in what appear to be primarily neurotic states. Nor, of course, do all depressives lose weight; on the contrary, some gain in weight, and even show a ravenous appetite, as did one of the cases studied here.

It is probable that enough has now been said to indicate that, for the manic-depressive psychoses, the "typical" case depicted by these criteria at best corresponds to a "type", but is not truly representative, and is not, therefore, really "typical". A much greater variety of manifestations than those suggested can be, and frequently are, shown; and the characteristics which are suggested are frequently not shown.

Necessity for broader conceptions of reaction types.—In other words, it is necessary both to modify and to enlarge the conception of what constitutes a manic-depressive type of reaction; and it is equally necessary to admit the presence of cases which present both manic-depressive "features", as well as those "features" which are recognized as being characteristic of neurotic states.

Briefly, this means that almost every conceivable mixture is to be found

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of the two frequently contrasted types of reaction, which, at present, are only distinguished for the purposes of clinical convenience.

Implications which do not follow.—This does not, of course, imply that all attempts at clinical groupings should be thrown overboard. Because the types of reaction which are recognized at present are also recognized as neither being clearly cut, nor accurately and rigidly differentiated and defined, it does not follow that they cease to have any meaning or use.

Nor does the presence of " every possibility of intermingling and no certain independence " between different types of reaction imply that attention should be concentrated upon the study of psychopathology as being more definite, to the neglect of the study of the clinical reactions produced, as being too vague. The recognition of the clinical reactions produced is just as important for purposes of correct assessment, and throws just as much light upon problems of prognosis and treatment, as does a study of the psychopathological factors which may have helped in their production. But it is important to recognize that clinical reactions can be extremely varied, and that " intermediate forms " frequently occur.

Finally, it may be well to point out that the conception of reaction types which has been put forward does not mean that genetic work in psychiatry must be a hopeless task. Genetic work in psychiatry is certainly rendered extremely difficult by the absence of clearly cut syndromes which, combined with the recognition of pathoplastic and other modifying factors, makes it extremely difficult to know what should be taken as representing the basic features of some sort of " biological entity " from which genetic studies can make a start. This is but to recognize the difficulty of the problem, and difficult problems can sometimes be solved.

Prognostic and Therapeutic Deductions.

Now the contentions which have been put forward have most important practical bearings. It will be remembered that the sharp differentiation between neuroses and manic-depressive psychoses was justified not only as being " in the interests of academic accuracy ", but also because of its " decisive bearing " upon questions of prognosis and treatment.

If the clear-cut diagnostic division is not always possible, so that certain cases cannot be definitely assigned into one category or the other, the application of the equally clear-cut prognostic and therapeutic deductions becomes, for such cases, an impossible task.

But a further source of trouble is that the prognostic and therapeutic deductions are not even true for many cases which can be so assigned.

It will be remembered that, in brief, these deductions were that, as regards prognosis, " the depressive is quite sure to become well ", but would probably relapse ; and that as regards therapy for the manic-depressive psychoses,

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" psychotherapy " should be avoided. This was because it was held that, although these cases run a course which is strikingly independent of environmental influences, yet this particular type of environmental influence could be actively harmful, which would be as unfortunate as it would seem to be contradictory.

The " neurotic ", on the other hand, did not recover unless active psychotherapy was employed, and, consequently, recovery could be directly attributed to psychotherapeutic intervention ; which makes the role of the physician differ in a most gratifying way.

Prognosis.

As papers which have recently appeared in this Journal (Anderson, Lewis) have clearly demonstrated, the conception of clear-cut recurrent episodes, with intervening periods of normal health, as being *the* course for the manic-depressive psychoses, is quite incorrect. Far from being quite " sure to become well ", the degree of recovery shown by patients who have suffered from definite depressive illnesses varies enormously, and quite a proportion never recover at all. Again, these illnesses not infrequently occur in individuals who have never enjoyed good health and, according to one investigation, about 50% do not relapse in such a way as to lead to hospitalization a second time (19).

The conception that continued ill-health, unless cut short by psychotherapy, constitutes the necessary course for the neuroses is equally erroneous. It is happily impossible to deny that neurotic reactions frequently show " spontaneous " recoveries and remissions, if by " spontaneous " is meant—most illegitimately—what cannot be directly attributed to intensive psychotherapy.

We must surely all have had experience of patients suffering from neuroses who give a history of a previous attack, not treated by intensive psychotherapy, and yet who, prior to the symptoms which lead them to seek medical advice, have enjoyed, after this previous attack, excellent health for many years. A case, not included in this series, which recently came under observation demonstrates these points in a striking way.

An unmarried woman, some years over 70, was referred because of gastric discomfort and nausea for which no physical cause could be found. It soon transpired that her symptoms had come on after she had been subjected to special and exceptional strains and difficulties not long before. Indeed there could be no doubt that she was suffering from a " neurosis ". Now this old lady had long enjoyed good health, and had made an excellent adjustment to a life which had been far from easy. But when she was in her early twenties, following upon a personal disappointment, she had become paralysed in all four limbs. She was finally admitted to a neurological hospital, where a

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world-famous neurologist had ordered that the "actual cautery" should be applied to her back. She made a rapid and dramatic recovery. Her disabilities had previously been present for several months.

Complete paralysis of all four limbs for which such drastic treatment had been ordered, and which cleared up so suddenly and completely as a result, could only have been hysterical in origin. Yet this patient, after this severe hysterical episode, which had been cut short by such vigorous methods, had remained apparently completely well for some fifty years. It is also not without interest that she had no realization whatever that her early illness had not been due to physical disease, and that she was far from being an unintelligent type.

It would indeed be hard to maintain or justify the belief that the innumerable people who become anxious and unhappy as the result of difficulties and disappointments, to the extent of becoming economically or socially handicapped, and in this sense definitely ill, and yet who recover, and remain perfectly well without ever consulting a physician, far less a psychotherapist, all really suffer from "psychotic" states. Yet, according to the view that neuroses do not recover, or remit "spontaneously", how else could their recovery be explained? And if the generalizaton is not meant to cover such "mild" cases, when does a case cease to be "mild"?

Psychotherapy.

(*a*) *Psychoses.*—It will be remembered that one of the authors quoted maintained that "melancholia was a psychosis, and it was *ipso facto* not amenable to psychotherapy. . . . He was referring to the word in its modern sense".

The first statement is of double interest. The generalization about "psychotherapy" is applied to all psychoses considered as a group, thence necessarily to the manic-depressive variety; and "psychotherapy" is evidently restricted to the description of at least relatively intensive and prolonged procedures, or some form of "analysis".

Now it would seem most unfortunate and unjustifiable arbitrarily to restrict the meaning of "psychotherapy" in this modern way, so that it becomes synonymous with intensive and relatively prolonged procedures, or some form of "analysis", particularly when by analysis is implied something approximating to the extremely lengthy (and costly) Freudian psycho-analytic technique. It is, of course, true that few would counsel the "analysis" of depressive states—certainly during their attacks. It is also true that intensive psychotherapy—(meaning by this, shall we say, not less than three personal interviews a week of not less than an hour each)—which would fall far short of what Freudians would admit to be an "analysis", may also appear capable of harm, and should be employed with great discretion and care. But there

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is a big and unwarrantable jump from such propositions to the proposition that no attempt at treatment, other than those directed against possible factors, should be practised at all.

It is put forward that many "manic-depressive" depressions are clearly modifiable, though in varying degree, by other than frankly physical means, or, in brief, by procedures which are psychotherapeutic. To employ these procedures is to employ psychotherapy, for it is impossible to imagine what else they can be. To give explanation, advice and reassurance, too often erroneously called "simple" by those who prefer the recondite, to order a holiday or arrange suitable occupation and recreation—these things may not be very dramatic, and therefore perhaps tend to be suspect; but that such procedures can be wonderfully effective in suitable cases all must, or should, admit.

(*b*) *Neuroses.*—It is equally certain that similar "simple" methods of treatment are all that are required for many cases at the neurotic end of the scale. Nor does this preclude the conviction that for many other cases intensive psychotherapy is absolutely essential if a good result is to be obtained. The case for intensive psychotherapy is, contrary to the assertion of some "psychotherapists", nowhere disputed to-day, certainly not by "psychiatrists". What is disputed is the view that the diagnosis of a neurosis implies the necessity for its administration, and that unless this is done, recovery cannot take place. It must also be remembered that for many "neuroses" intensive psychotherapy is of little avail, and that attempts at management is all that can be done. "Let us remember," writes Adolf Meyer, "that many neuroses are far more problematic and difficult to treat than the frank psychoses" (9).

It is therefore held that it should not be necessary to regard as reactionary obscurantists those who do not propose "intensive psychotherapy" or analysis for every neurotic case they see; nor as ignorant optimists those who in a real sense attempt to treat, and not merely to guard, the depressive ones.

Conclusion.

The main conclusion which forces itself upon attention is the prevalence of the desire for simplicity which finds expression in the adoption of that "either/or" attitude against which Adolf Meyer has so long inveighed, and which has wrought such evil in psychological medicine. We all of us agree that physical and mental are not mutually exclusive alternatives, and that very frequently the question "Physical or mental?" only acquires meaning if prefaced by the query "To what extent?" But we do not all agree that in making the diagnosis of "neurotic" or "psychotic" the question to be asked should often be of a similar kind. This is particularly unfortunate when the treatment to be given is thought to depend in the most categorical

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manner upon the answer obtained. Our problem should not be, Is this case neurotic or psychotic? with the deduction that if the former, then psychotherapy should be employed, and if the latter, it should be avoided; but rather, To what extent and in what way can this reaction be modified by "mental" as well as by "physical" means?

"It is obvious," writes Ross (20), "that the man who believes neurosis and psychosis are much the same thing will not make a vigorous attack on either illness". But this is to make a belief in continuity imply identity, and all the members of a *graded* series, by definition, are not the same. Most psychiatrists would advocate a judicious vigour in the treatment of all their patients untrammelled by rigid rules which seem unjustified generalizations for special groups, rather than fitting all the facts to be observed.

It can at least be hoped that much of what has been discussed will soon concern the medical historian alone, and that rigid formalism will lose its appeal; that psychiatry will not be separated from general medicine and will not be confined to that impossible category "the mind", and that its subject-matter will not be divided into psychoses and neuroses as sharply separated as the psychiatrist, who should alone deal with the former, and the psychotherapist, who should alone deal with the latter, each in his different way.

Summary.

It is pointed out that divergent, and indeed contradictory, opinions have been expressed as to the possibility of dividing those cases which show the various manifestations of anxiety and depression as the outstanding features into two contrasted categories — (1) neuroses, and (2) manic-depressive psychoses.

Some claim that a sharp division is not only possible, but extremely desirable, because of the prognostic and therapeutic implications; whilst others affirm there is a "continuous and graded series", and their views on prognosis and therapy suffer from a corresponding lack of definition.

Certain criteria have been selected from amongst those which have been put forward as achieving this differentiation, and an attempt has then been made to apply these criteria to a group of cases which have been personally observed. As a result of this attempted application, no sharp division appears to be achieved. It is argued that it does not in fact exist, and that its attempted erection results from a lingering formalism based upon the conception of "disease entities"; whereas the broader conception of "reaction types" fits the facts to be observed in a more satisfactory way.

If this distinction does not exist, the prognostic and therapeutic implications based upon this distinction become suspect; and it is put forward that they are unjustified generalizations, and can only be held to apply to very small groups. Special exception has been taken to what seems an undue

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restriction of the meaning of psychotherapy to some form of "analysis", which is coupled with an undue extension of the necessity for its application as used in this restricted sense—a view which does not preclude the conviction that "intensive" psychotherapy is often called for. Finally it is put forward that the erection of a sharp division between the neuroses and the manic-depressive psychoses is an example of that tendency towards psychiatric dichotomies which is to be deplored.

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THE ENUMERATION OF THE BLOOD-PLATELETS IN MENTAL DISORDERS.*

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Introduction.

On looking through the literature relating to the blood-picture in various types of mental disorders, one finds that most workers have confined themselves to the red and white cells only. Relatively little attention has been given to the study of the third element of the blood, namely, the blood-platelet. It is now almost universally accepted that the blood-platelet is a constant element in normal blood, and it has been shown both clinically and experimentally that in health and in disease the platelets fluctuate independently of the red and white cells. In toxic conditions there is agreement that the platelets fall in the acute stages, and that during convalescence their numbers tend to increase, after which there is a gradual fall to the normal limits. This constant behaviour of the platelets has been observed in certain acute infectious diseases, in pneumonia and in typhoid fever. Certain writers think that the platelets have an important function in eliminating foreign bodies from the blood, or play some part in establishing a state of immunity to infection. This part played by the platelets in the sterility of the blood-stream suggests an explanation for the reduced numbers in acute infections. Diminution would be due to the using-up of the platelets in removing and destroying the infective agents or their products. The present work gives the results of an attempt to show that this same relation between the platelets and toxins also exists during certain phases of mental disorder and, in these particular cases, a toxin from a focus of infection can be assumed to be the causative agent in producing the psychosis.

Historical.

The existence of the platelet as a third morphological element of the blood has been repeatedly affirmed and denied. The earliest observers were Donne and Arnold (1) about the middle of the last century. Bizzozero (2), who gave the platelets their name, described them as they appeared in the mesenteric

* An essay for which the Bronze Medal of the Royal Medico-Psychological Association was awarded, 1936.

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vessels of the guinea-pig and his observations were confirmed by others, notably Ebert and Schimmerbusch (3). Marino (4) failed to observe them in blood which was allowed to flow into absolute alcohol, but Downey (5) showed that 100% alcohol readily distorted the platelets and rendered them difficult to stain and demonstrate. Many theories have been advanced to explain the origin of blood-platelets, and the theories which hold that the platelets are primary forms of red blood-corpuscles have long been discarded. Schilling (6) believes that the blood-platelet itself is the entire modified nucleus of the younger erythrocyte, partly detached and only becoming free in the circulation, e.g., by blood-platelet thrombosis. The same author concludes that they are not fragments of leucocytes because specific granules are always absent, and that the theory that they are pieces of erythrocytes is contradicted by the fact that hæmoglobin cannot normally be demonstrated. In his classical work Wright (7) has demonstrated that platelets are portions of megakaryocyte cytoplasm of the bone-marrow which, after segmentation, are detached and carried off in the blood-stream. In support of his theory Wright has pointed out the parallelism which exists between the number of platelets in the circulation and the number of megakaryocytes in the bone-marrow. In pathological states megakaryocytes have been found in the spleen, lungs and blood stream, and they are present in all stages of development in the bone-marrow. The youngest cells are non-granular and have a single nucleus, which matures to become larger and more complex, until ultimately several nuclei are formed (8). The cytoplasm also increases in size and becomes full of fine granules, which Wright (7) and Downey (9) consider peculiar to the platelet and the megakaryocyte. Thus the fully-developed cell is very large and has a greater cytoplasm containing a number of nuclei. It is only in the adult stage that megakaryocytes can produce platelets. Wright also points out that platelets do not appear in the embryo before the appearance of the megakaryocytes, and that they are only found in mammals in which megakaryocytes are present. Platelets and megakaryocytes are both increased in the blood following toxæmia, inflammatory states, and in experimental lesions following the injection of anti-platelet serum (10), or of diphtheria antitoxin (11). Both are diminished in pernicious anæmia (12) and after exposure to X-rays (13).

From the foregoing it appears that the platelet normally arises from the megakaryocyte of the bone-marrow. Brown and Bunting (14) assume that in time of stress platelets may arise from mononuclear and transitional cells of the bone-marrow, spleen and glands.

The term " thrombocyte ", introduced by Dekbuyzen to describe the spindle-shaped cells found in the blood of amphibians, is now frequently used for all elements of the blood which are smaller than erythrocytes. It includes the blood-platelets of mammals and the small nucleated corpuscles of birds and the lower vertebrates (15).

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Destruction of Platelets.

Bedson (16) concludes that the younger cells of the reticulo-endothelial system, of which a large number are situated in the spleen, are normally responsible for the removal of platelets from the blood, and he also reports an increase after splenectomy. In health the spleen contains a large number of platelets, and in disease it has been found to contain a relatively large number of these elements when their numbers are diminished in the blood (17). Mackay (18) concludes that the life of the platelet is short and that a large number are produced and destroyed each day, while Duke (19) considers that circulating platelets degenerate within three to five days.

Numerical Variation of Blood-platelets in Health and Disease.

Cacurri (20) reports that after exercise there is a diminution in the platelet-count lasting a few hours only, due to fatigue liberating harmful products. No change was observed during sleep or lying down. Wittkower (21) notes an increase after a hot bath.

In disease, variations are more marked than in health, and occur during a single illness, according to the stage, whether acute or convalescent. Furthermore, the behaviour depends on the reactive power of the bone-marrow. It can be stated in a very general way that platelets are diminished in leukæmia, acute tonsillitis, lobar pneumonia (pre-critical stage) and pyæmia. They are normal or very slightly increased in scurvy, diabetes mellitus and hyperthyroidism, while in secondary anæmia (post-hæmorrhagic), lobar pneumonia (convalescence), acute rheumatism, nephritis, malignant disease and chronic forms of tuberculosis they are normal or increased (22). The platelets show a rise following parturition and after operation (23).

In acute cases of toxæmia there is a tendency for the platelets to be diminished, and during convalescence the numbers tend to increase, after which there is a gradual fall to the normal limits. This constant behaviour of platelets has also been observed in many acute infectious diseases, e.g., in diphtheria (26), the common cold (27), and smallpox (27*a*). The most comprehensive observations on the platelets in a typical acute infection are those of Reimann (24). He found that in the majority of a series of cases of pneumonia the platelets were considerably reduced during the pyrexia, and began to rise shortly after the crisis, reaching a maximum in from 6–9 days and returning to normal in a further similar period. He further noted that when complications occurred there was a further drop in the platelets. Kemp, Colhoun and Harris (25) state that during the course of typhoid fever the number of platelets is normal or subnormal. "If the fever breaks by crisis, there is a rapid and striking rise in the platelets. If this rise does not occur it suggests a masked serious complication." Duke (26), describing experiments

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with diphtheria toxin, killed typhoid bacilli, X-rays and benzol, found that while all these depressed the platelet count, in small doses they frequently caused an elevation.

Certain writers think that the platelets have an important function in eliminating foreign bodies from the blood, or play some part in establishing a state of immunity to infection. Bull (29), in a series of publications, reports that streptococci and pneumococci, unless extremely virulent, were rapidly removed from the blood of experimental animals. He further observed that typhoid and dysentery bacilli of the Flexner group were agglutinated in the blood stream. In both cases the agglutinated organisms were rapidly removed and destroyed in the visceral capillaries. Any bacilli not so agglutinated remained in the blood and produced a septicæmia. Dalrez and Govaerts (30), following up the work of Bull, demonstrated that, in the blood-stream of a rabbit, staphylococci and *paratyphosus B* bacilli adhered to the platelets. This adhesion was associated with the disappearance of bacteria and a diminution of the number of circulating platelets. Two minutes after injection mixed masses of platelets and organisms were found in the capillaries of the spleen and liver. Continuing these investigations, Govaerts found that intravenous injections of washed platelets accelerated the elimination of typhoid bacilli from the blood.

All the above work suggests that the platelets play a part in the sterility of the blood-stream, and it also suggests an explanation for the reduced numbers in acute infections. Diminution would be due to the using up of platelets in removing and destroying infective agents. Dawburn, Easlam and Evans (31), summarizing the literature on the effect of sepsis on the platelet, report : " There is a very definite measure of agreement as to the fall in the acute stages and the rise in convalescence on the platelets in acute generalized infections."

Focal Infections and Mental Disorder.

For years past intensive investigations have been carried out on the study of nerve disorders based upon alterations in the blood and glands, and from the results of these it is realized that infection plays a large part in the causation of mental disorder (Hunter (32), Cotton (33)). Mapother (34) reports Cotton as saying : " Mental disorders are due to a combination of many factors, but the most constant one is the intracerebral biochemical cellular disturbances arising from circulating toxins originating in chronic foci of infection situated anywhere throughout the body, associated probably with secondary disturbances of the endocrine system."

Moynihan (35) reported cases of mental disorder relieved by operation for chronic appendix, gastric ulcer and gall-bladder. He concluded that the infective agencies which appear sometimes associated with mental diseases are the teeth, the facial sinuses, the alimentary tract and gall-bladder ; and

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that infections, wherever arising, are apt when they reach the cæcum and ascending colon to remain there long after the primary focus has ceased to be active. His work shows that mental instability and insanity may sometimes be relieved, and apparently cured, by the eradication of a focus of infection, or the removal of the disease that it has originated.

Graves (36) stresses the frequency of sinus infection, and claims that the treatment of these conditions is followed in a very large number of cases by physical and mental improvement, even in cases that have been in a stationary or progressive phase of mental deterioration for so long as to be considered hopeless.

Watson-Williams (37) has shown that a septic focus in the teeth, tonsils, prostate gland or gastro-intestinal tract may be the cause of remote systemic disorders, and the work of Rosenow has been of great value in helping to explain the mechanism of these processes. Rosenow speaks of the elective localization of micro-organisms, and this may be decided by qualities of the soil no less than of the seed.

Clarke (38), though not entirely confirming Graves's results, concludes that focal sepsis can predispose to, and exaggerate the development of, mental symptoms; infections of the ear, nose and throat are likely to be particularly potent in doing so; the incidence of ear, throat and nose infections in the insane is considerable.

Many important psychiatrists have called in question the results claimed, and the alleged "proven relationship" of sepsis and mental disorder, chief among whom are Henderson and Menzies. Henderson challenges the findings of Hunter and Graves. He states (39) that Kopeloff and Kirby examined 120 patients, dividing them into two groups as nearly identical as possible. One group was treated surgically, while the other group had no surgical treatment. The percentage recovery-rate of the manic-depressive patients in the two groups was equal, while patients with dementia præcox showed a slightly lower recovery-rate in those operated upon than in those left alone.

Menzies agrees that the incidence of chronic sepsis is no more frequent in the insane than in the sane (40), sepsis is, as a rule, a secondary cause of mental disorder, the recovery-rate has not been materially raised by antiseptic measures, the use of autogenous vaccines has proved disappointing, and serological, metabolic and endocrinological tests have all given results that are not at all specific. However, it is not denied that the end-result from severe attacks of septicæmia may be mental disorder.

The mode of action of toxins in the causation of mental disorder is conveniently reviewed by Ford Robertson (41):

"If a toxin is circulating in the blood, it will fix itself on any substance for which it has a chemical affinity. There are many toxins, and some of them of bacterial origin, that have a special affinity for nerve-cells. The liability to be injured by such neurotoxin is, however, not the same in all nerve-cells.

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There are wide local and individual differences dependent upon congenital and acquired qualities. Special vulnerability to toxins on the part of nerve-cells of the association centres in some stocks appears clearly to be at least one important factor included in hereditary predisposition to insanity. Two distinct effects upon the association centres must be recognized. During the height of the toxic action the metabolic processes occurring in these centres are seriously disturbed: mental reactions are correspondingly disordered, and consequently there is confusion, excitement, depression and stupor. If the toxic action subsides, there may be complete recovery on the part of the tissues, in which case the mental reactions again become normal. In other cases there is irreparable destruction of many neurons, and the centres are permanently damaged; the mental reactions, therefore, remain more or less abnormal. Thus regarded, mental disorders are abnormal reactions of an associative mechanism that is damaged by active, or former, disease, or by traumatism, or that is defective owing to some developmental fault."

From a consideration of the literature and clinically and experimentally there is a great deal of evidence in support of the contention that focal infections are capable of causing serious mental disturbance, and that they cause the most serious effects at a distance.

Method.

It is to be regretted that there is no standard method of counting the blood-platelets. With each technique different counts are obtained, and some workers consider the method which gives the highest figures to be the most accurate. Platelets are easily damaged; they can be disintegrated by the pressure of a cover-slip; they also clump very easily, either to each other or to the red blood-corpuscles. It is of the utmost importance to employ methods that do not damage the platelets and also prevent their clumping.

Throughout the work reported upon in this paper I have used the method described by Cumings (42). By this technique the results are more accurate and stable, the platelets are evenly distributed over all the fields, they show no evidence of clumping and they are not damaged in any way because their shape, size and outline are distinct. By other methods I found counting was rendered extremely difficult, owing to mass clumping of platelets, while in stained films platelets adhering to red cells and clumping gave rise to inaccuracies in the results.

Cumings claims that the absence of clumping in his method is partly the result of using paraffin containers, also that small platelets are not damaged but well preserved owing to the addition of mercuric chloride to the diluting solution. Using this method Cumings's findings conform to those of Flossner and Dameshek Olef, who obtained figures from adult males of about 600,000 per c.mm.

The actual technique employed is as follows:

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Two solutions are required :—

Solution A :

Sodium chloride	0·8%.
Calcium chloride	0·02%.
Potassium chloride	0·02%.
Sodium bicarbonate	0·1%.
Sodium acid phosphate	0·005%.
Magnesium chloride	0·01%.

To prevent contamination, add to above solution mercuric chloride 1/10,000.

Solution B :

1% mercuric chloride in normal saline.

Immediately before use add 5 parts of solution A to 1 part of solution B, without filtering either fluid. Blood is obtained from the ear by pricking through a drop of diluting fluid, without using local pressure, and allowing the blood to flow directly into the diluting fluid, which is in a small paraffined bowl (about 1 in. × ½ in. × ¼ in.). By means of a paraffined rod, or better, by means of a piece of glass tubing coated within and without with paraffin, through which air can be blown by means of a rubber teat, the contents are thoroughly mixed. The exact degree of dilution is that which, by experience, is shown to give the greatest enumeration—usually about 1–400. The ear is carefully dried and blood taken for a red corpuscle-count, which is performed in the usual manner.

By means of the paraffined glass mixer a drop of the diluted blood is placed on a counting chamber, and the cover-slip applied. After the red cells and platelets have settled, usually in ten minutes, the platelets are counted against the red cells, a thousand of the latter being enumerated, using a high power eye-piece (× 8) and a dry lens (⅛ in.). It is convenient to have about 150 red cells in a field, and the original dilution of the blood should be such as to give this figure. The count should always be performed within half an hour of collection. The counting chamber should be kept specially for platelet counts, and very carefully cleansed before use; if found scratched it must be discarded. A drop of the diluting fluid should always be examined each day before starting to see that no free particles, such as might resemble platelets, are present.

The platelets are easily recognized as highly refractile particles, varying in size from about one-eighth to one-half the diameter of a red cell. Their shape is round, oval or oblong, with well-defined edges, and they show no movement once they have settled in a counting chamber.

The results to be described are of male cases only; the patients chosen were mainly new admissions, except for a number of cases of schizophrenia; all were under 55 years of age and all were free from intercurrent disease, thus eliminating the fallacies that might be due to menstruation (in the female), to diseases of the blood and organs, especially arterial degeneration and nephritis, and to senility. The examinations were performed at the same time each morning, four hours after breakfast, and no medicine was administered for twelve hours previously. The patients were kept resting in bed all the morning, so that no fallacy could arise from fatigue and exercise.

The cases—95 in number—will be subdivided into groups, which will be considered separately.

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MANIC-DEPRESSIVE INSANITIES.

Twenty-eight of the patients come under this category, namely, 17 maniacs, 7 melancholics and 4 cases of alternating insanity.

Examples of four cases of mania will be given first, as in these cases a blood-platelet enumeration was performed each day during the full course of the acute illness.

CASE 1.—T. W—, aged 53, admitted August 23, 1934. Weftman, married.

Past history.—Fourth attack; previous attacks 1921, 1923 and 1927. No illnesses, except influenza and "chronic sniffles" during the winters. Wife and friends noticed that he was getting sleepless and restless for two weeks.

Family history.—Negative.

Condition on admission.—Temperature 98·4° F., pulse 80, respirations 20. Pale, rather toxic looking, but colour of membranes and lips good; edentulous (all teeth extracted when last certified and in this hospital; at that time had pyorrhœa, and teeth were carious). Gums apparently healthy and show no evidence of gingivitis. Tongue furred. Chest and abdomen: nothing abnormal found. Blood-pressure 120. All reflexes normal.

Wassermann, Meinicke and Widal tests negative.

Urine: S.G. 1024, acid, negative for sugar and albumen.

Mental condition.—Admitted in an acutely maniacal condition. Was greatly excited and generally uncontrolled. He was distractible, and his attention could only be sustained for a few seconds. Talked incoherent nonsense, switched from one subject to another and, at times, shouted, sung or cried in turn. Very restless, mischievous, and inclined to be destructive and depraved in his habits.

Progress and course:

August 24: Continually shouting and talking nonsense. Required to be under special supervision. After paraldehyde 2 drm. slept for five hours.

August 25: Uncontrolled in his thoughts and actions. Maintains his excitement and restlessness. Slept little.

August 26: Restless and excited behaviour continued.

August 27: Takes his diet when spoon-fed. Manifests distractibility and "flight of ideas". Slept five hours overnight. Still requires special supervision.

August 28: Given hypodermic of morphine and hyoscine; slept for five hours.

August 29: Patient in a condition of stupor; is more settled in his behaviour. Taking diet and sleeping better.

September 1: Has gained control over himself and is more rational in his talk and conduct. Slight amnesia and confusion for the events of the past few days.

September 3: Well orientated; realizes that he has been ill mentally. Conversation normal and attention good. Sleeps well now.

Date.	Erythrocytes per c.mm.	Platelets per c.mm.
August 24	6,000,000	410,000
,, 25	6,200,000	410,000
,, 26	6,250,000	420,000
,, 27	6,000,000	430,000
,, 28	6,000,000	425,000
,, 29	6,200,000	300,000
,, 30	5,500,000	550,000
,, 31	5,750,000	520,000
September 1	5,500,000	620,000
,, 2	5,750,000	600,000
,, 6	5,600,000	600,000

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CASE 2.—L. T—, single, aged 21, motor driver, admitted January 22, 1934. First certification.

Family history.—History of intemperance on mother's side.

Personal history.—Suffered from no illnesses except common colds and bronchitis since childhood.

Present illness.—Very sudden onset. Was apparently well until the day previous to admission.

Physical state.—Temperature and pulse normal. Tongue coated and tonsils inflamed. Teeth fairly good. Nothing abnormal in chest and abdomen. Reflexes normal.

Urine: S.G. 1020, acid, no abnormal constituents.

Wassermann, Meinicke and Widal negative.

Mental state.—Wildly excited, noisy, resistive, distractible, rambles incessantly and incoherently in his talk.

Progress and course.—For four days his excitement continued, and injections of morphine and hyoscine had to be administered.

January 26: In condition of stadium debilitatis. Knows all that is going on around him, amenable, manageable.

January 29: On the whole much better. Realizes that he has been ill and his mental symptoms have disappeared.

February: Made satisfactory progress and was discharged recovered.

Date.	Erythrocytes per c.mm.	Platelets per c.mm.
January 23	6,000,000	300,000
,, 24	5,600,000	260,000
,, 25	5,700,000	500,000
,, 26	6,200,000	540,000
,, 27	6,000,000	500,000
,, 28	5,700,000	690,000
,, 29	5,500,000	600,000
,, 30	5,700,000	625,000
February 9	5,750,000	635,000

CASE 3.—S. C—, male, aged 35, chimney sweep, married. First certification.

Family history.—No history available.

Physical state.—Fairly well nourished. Normal pulse. No gross lesions of the major viscera. Advanced pyorrhœa and teeth carious.

Urine normal. Wassermann negative.

Mental condition.—Rambling and incoherent in his statements, which follow each other with extreme rapidity. Exalted, with delusions of persecution. Restless and mischievous.

Progress and course.—For four days after admission had to be confined to a side room and required special supervision.

January 31: Extreme symptoms subsided, was somewhat stuporose, but was amenable and controlled in his conduct.

February 2: Showing more interest, association of ideas normal, well orientated and has insight.

Maintained a steady progress.

Date.	Erythrocytes per c.mm.	Platelets per c.mm.
January 28	5,800,000	300,000
,, 29	6,000,000	420,000
,, 30	6,000,000	450,000
,, 31	5,500,000	500,000
February 1	5,800,000	520,000
,, 2	5,500,000	600,000
,, 3	5,600,000	700,000
,, 4	5,600,000	610,000

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CASE 4.—G. M—, single, aged 25. First attack.

Has chronic rhinitis. Anæmic and toxic-looking on admission. No evidence of organic disease, and urine and blood examination negative.

Mental state.—Uncontrolled in thoughts and actions for four days. Afterwards became manageable and amenable, and in seven days had recovered from his acute symptoms. Was discharged recovered.

Date.	Erythrocytes per c.mm.	Platelets per c.mm.
March 3	5,700,000	460,000
,, 4	6,000,000	460,000
,, 5	5,800,000	500,000
,, 6	5,500,000	500,000
,, 7	5,750,000	550,000
,, 8	5,700,000	610,000
,, 9	5,750,000	610,000

COMMENT ON CASES.

The tables show the results obtained in these four cases. On examining the figures it is clear that on the days after admission there was a marked diminution in the circulating platelets of the blood, and that the count remained at a low level during the four days in which the patients were uncontrolled and the mental symptoms were most acute. In Case 1 the count was stationary during the acute phase, and in Cases 2 and 3, although there was a slight increase noted each day the numbers did not reach normal limits. On the next three days, while the patients were in the "stadium debilitatis" there was a rapid rise in the platelet numbers, which increase rose to normal on the third day of this stage. On the first day of the convalescence the platelets exceeded the normal numbers for the one day only, after which they fell to normal and remained at this level.

It is reasonable to assume that in these four cases a toxic influence was the causative agent in producing the rise and fall in the circulating platelets. These findings are conformable to those reported in fevers and generalized toxic conditions, except that, in these four cases of mania, the increase during convalescence lasted the one day only, whereas in generalized toxæmia the increase in the platelet numbers at this stage is stated to last for a number of days. This could be accounted for by the larger amount of toxin absorbed into the blood during the acute stages of the more generalized illness making a greater call upon the defensive forces of the blood and, during the convalescent stages, the activity of the hæmopoietic tissues will necessarily have to be prolonged to restore the blood elements to the normal numbers. That a chronic focus of infection was existent in these four cases is evident from the histories. In Case 1 the patient has had attacks of influenza, and during the winter months suffered from chronic "sniffles", Case 2 suffered from chronic bronchitis, in Case 3 the teeth were carious and the gums showed pyorrhœa, and in Case 4 there was chronic rhinitis.

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From a consideration of these cases it is apparent that it was during the "stadium acutum" that the neurotoxin from a focus of infection acting on the cortical cells and causing the cerebral irritation was greatest in its intensity, and that the platelets acting as agents in eliminating foreign substances from the blood were being used up, and were, accordingly, reduced in numbers. In the exhaustive stages, and with the subsidence of the acute symptoms, the rise in the platelet numbers shows that the toxæmia has been counteracted, and the platelets, not being used up in neutralizing the toxin, begin to return to the general circulation. The increase during the first day of the convalescence was merely reactionary on the part of the bone-marrow, and during convalescence the toxin has been completely eliminated and the platelets are therefore found in their normal numbers in the drop of blood examined.

Another feature in favour of the contention that there is a toxin present in the blood in the cases of mania was noted during this investigation. Platelets are generally described as varying in shape and size, some being half the size of a red blood-corpuscle and rounded or pointed in shape, but in the acute stages of the maniacal attack no large or intermediate platelets were observed, all being small, rounded, with well-defined edges and about one-sixteenth the size of a red cell. This would appear to be due to the larger platelets being used up, the observed smaller forms being the new forms from stimulation and response of the bone-marrow. The larger forms returned when the platelet-count began to rise, and during the convalescence their shape and size was normal.

In two other cases of mania, in which the acute symptoms lasted three or four weeks, no fluctuation in the platelets was noted, and they were normal in numbers and appearance until two to three days before the patients became controlled and amenable; then the numbers decreased, small platelets predominating, and they remained depressed until the subsidence of the excitability, when the platelets returned to their normal numbers.

It will be seen from a consideration of the above two cases that the symptoms did not subside until three days after the platelets showed a decrease. It would appear that there was an inability on the part of the platelets from the beginning of the illness to neutralize the toxin, which, by remaining active, caused the acute symptoms. When the platelets overcame the inhibiting action of the toxin, as shown by their reduced numbers and the preponderance of the small forms of platelets, the patients improved mentally.

Four cases of mania which died within a few days of admission only allowed of a platelet-count on the first and second days after admission. In three of these cases the count was decreased, being under 500,000 on the two days, and in the other case the count was 550,000 on the first day, and on the second day, a few hours before death, the numbers rose to 1,000,000.

Owing to the necessity of having to administer sedative drugs to three cases, a count was only possible at irregular intervals, but in these three cases

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a decreased count was noted when the blood was examined during the excited phase of the illness.

Normal or slightly increased counts were only found in three of the cases of mania examined.

In one case, which had septic abrasions on his hips, thighs and both arms, caused in a cycle accident four days prior to admission, an increase was noted, but when the patient returned to a normal mental stability and his septic wounds had healed a normal count was recorded.

ACUTE MELANCHOLIA.

Case.	Platelet-count on admission.	Average platelet-count during acute illness.	Duration of acute illness.	Platelet numbers on recovery.	Remarks.
A. S—	390,000	450,000	18 days	650,000	Recovered.
W. B—	400,000	500,000	14 ,,	600,000	,,
F. B—	400,000	400,000	12 ,,	550,000	,,
P. L—	420,000	480,000	28 ,,	625,000	,,
A. S—	800,000	700,000	Died six weeks after admission.		
R. N—	760,000	700,000	6 weeks	650,000	Recovered.
R. B—	700,000	700,000	8 ,,	700,000	,,

In this group 7 cases of typical melancholia were taken and blood-counts were performed on every third day during the acute illness of the patients. From a consideration of the above table it will be seen that in four cases a decreased count was obtained on admission, and in all during the acute illness the platelet numbers remained low. On recovery the platelet numbers returned to the normal limits and no increase was noted during convalescence. In two of the remaining cases the number of platelets was slightly above the normal, and in the seventh case the numbers remained at a stationary level throughout the illness.

It is interesting to know that in the four cases in which a low platelet-count prevailed during the acute stages of the illness, the depression and active symptoms subsided sooner than in those cases in which the platelets remained high, and it can be considered a reasonable assumption that in these cases a toxic factor was present, and that the platelets, by being reduced, were counteracting the toxin.

ALTERNATING INSANITY.

Firstly I will submit one case as a typical example of this group :

G. E. C—, aged 42. Very unstable and unreliable from 28 years. Has periods of depression alternating with attacks of mania. Shows remissions lasting 1–3 months. During his excited phase he is generally uncontrolled and impulsive and, when depressed, is sullen, morose, self-absorbed and seldom speaks.

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Platelet-count per c.mm.

During excited phase :		
1st day	350,000.	Uncontrolled.
8th ,, ...	500,000.	Beginning to settle.
12th ,, ...	600,000.	Controlled and rational.
During remission :	650,000.	
During melancholic phase :		
1st day	400,000.	Profoundly depressed.
10th ,, ...	625,000.	Brighter.
18th ,, ...	700,000.	Depression subsided.

In three other cases in this group findings conforming to the above were found, and in these cases the numbers on the day of relapse were 420,000, 420,000 and 440,000 respectively. With the relieving of the symptoms the platelets gradually rose until they were normal or within normal limits when the patients had regained temporary stability.

Primary Dementia.

In this group twenty cases were investigated, and normal readings or a slight increase in the numbers of the platelets were noted.

Delusional Insanity, Chronic Mania and Chronic Melancholia.

No variation from the normal numbers was observed in these groups.

Dementia : Senile and Secondary.

These cases gave normal findings except in three instances, which showed a marked increase during exacerbation of the mental symptoms. As these three cases could not be followed up no definite conclusions can be arrived at.

Dementia Paralytica.

Ten cases suffering from this condition were examined. The most significant feature was that an altered type of platelet predominated. This was large, irregular in shape and darker than the platelet seen in normal blood. In a general way it may be stated that the more progressive and advanced the disease the more large and irregular platelets were observed.

It is now accepted that many of the symptoms of general paralysis of the insane are caused by the toxic agent produced by the spirochæte, and there can be no doubt that the toxin is acting on the blood and tissues from shortly after the patient has contracted the disease. Schilling reports (43) an increase in the number of platelets in syphilis. All the cases of dementia paralytica reported above gave a history of having been infected for more than ten years previous to admission. This would mean that during all these years the

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platelets were increased, were continually being destroyed and that, after a time, the strain could no longer be borne by the platelet-forming tissues and, in consequence, these tissues show exhaustive or other changes. In pernicious anæmia, Gulland and Goodall (44) consider that the abnormal red blood-cells seen in the blood are due to a toxic effect on the bone-marrow, causing a degeneration which may be looked upon as a reversion to the fœtal type of marrow. It is reasonable to assume that in dementia paralytica, as in pernicious anæmia, the constant drain on the platelet-forming tissues, as a result of the prolonged action of the syphilis toxin, leads to a fœtal type of marrow and, consequently, the appearance of the large irregular forms of platelets in the circulating blood. This contention is strengthened by the fact that Schilling (43) reports that in childhood the platelets are increased in size, and Wright (7) has demonstrated that the younger cells are non-granular and become larger on maturation.

General Conclusion.

Valentine (45), in his experiments with staphylococcal infection on human and rabbit blood, concludes that " the comparatively poor development of circulating antihæmolysin in many patients suffering from chronic furunculosis may be due to the fact that the toxin is fixed locally in the tissues and does not therefore stimulate a general immunity. In a deeply-seated abscess the absorption to toxin appears to be easier ". It is my opinion that the platelet response to toxins is the same as that of antihæmolysin to infection. I conclude that in the cases of mania, melancholia and alternating insanity which showed a fluctuation in the circulating platelets conforming to that shown in generalized toxic conditions, a circulating toxin was operative and caused the psychotic symptoms, and that the toxin arose from deeply-seated abscesses. Graves stresses the frequency of deep sinus infection in most of his cases, and it will be observed that the cases of mania quoted in this paper show evidence of deep infective processes.

In all the other cases of mental disease reported upon, and in which no fluctuation was noted, a focus of infection may have been present, but the toxin from this may have been fixed locally and caused no reaction in the circulating blood, or the toxins, having been acting over a fairly long period, may have established a general immunity in the patients and, therefore, no change in the blood could be expected.

Summary.

1. In four cases of mania the platelet fluctuation conformed to that observed in generalized toxic conditions. In these cases a toxin from a focus of infection may be assumed to be present.

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2. While the platelets remained at the normal level in two cases of mania, there was no change in the mental symptoms, but when the number of platelets fell, indicating a possible neutralization of toxins, the acute mental symptoms subsided.

3. In the cases of mania only small forms of platelets were observed. This would appear to be due to the larger forms being used up in counteracting the toxin, the smaller forms being the new platelets from bone-marrow response.

4. Four cases of melancholia which showed a low platelet count recovered more quickly than cases which showed no platelet response.

5. During the relapse stage in alternating insanity the platelets diminished, probably indicating a body response to a toxin.

6. No variation in the platelet numbers were noted in chronic forms of mental disease.

7. In dementia paralytica a large, irregular type of platelet, darker than normal, predominated, and the more advanced the disease the more these large irregular forms were seen in the films examined. This is probably due to a degeneration of the bone-marrow, from the prolonged action of the syphilis toxin, causing a reversion to a fœtal type of marrow.

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A CASE OF HYSTERIA SHOWING SPONTANEOUS HYPERVENTILATION TETANY.

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THE condition of spontaneous hyperventilation tetany has received some attention recently, and in many cases it has been impossible to demonstrate adequate biochemical changes to account for the tetanic manifestations, which so closely resemble the more recognized clinical types of tetany. The reason for the apparent spontaneity of the attacks has remained obscure beyond the general trend to recognize it as " functional " in most cases. In view of this recent interest, and of the atypical clinical manifestations in this case, the following observations on a single patient are put on record. A few previous cases treated at the Maudsley Hospital, but differing from the present one in the absence of loss of consciousness during the attacks, are also briefly reported.

CASE-HISTORY.

The patient, a probationer nurse, aged 20, was admitted to the Maudsley Hospital because of frequent fits and other symptoms diagnosed as hysterical.

Family history.—Both sides of the family show unstable nervous tendencies, including heavy drinking on the father's side. There had been much family discord all her life ; her father and mother being separated, she, the youngest of four siblings, lived for varying periods with each parent.

Past history.—Infancy and the milestones of development were normal. She had low average results at school, which she never liked, and where she finished in standard 7, at the age of 14. Since school she has had four different positions, each lasting about a year till she became dissatisfied.

Previous illnesses.—At the age of 10 months she had whooping-cough, during which she had fits apparently similar to the present ones ; they started suddenly on the third day and then recurred at two-hourly intervals for the next three days, during all of which she was blue, and appeared unconscious. She recovered, but had frequent colds subsequently, and, till the age of 7, similar fits about once a year, followed by bouts of bronchitis with cough and temperature.

At the age of 15 she was admitted to a hospital for persistent cough, which had lasted for months, and pain in the right iliac fossa similar to that which she had with her periods. Appendicectomy was performed ; during the second week after the operation she became restless, had retention of urine, and her wound reopened (due to her interference, it was thought). She was diagnosed as chorea and sent to a convalescent home, from which she was able to return to work after 3 months.

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Menstrual history.—Her menses started at the age of 11 ; her periods were regular (7/28), with a rather heavy loss and pain in the right iliac fossa. After the appendicectomy her periodicity was 7/35, with more loss (2–3 diapers per day), and she had leucorrhœa. With her periods she has always had headaches and faintness on stooping.

Personality.—As a child she was " such a little monkey " and never still. She was obstinate and bad-tempered, and consequently had few friends, though wishing for them. She was reserved and would day-dream a lot. Her mother was " the only one who understood her ". She had few interests, but during the last four years had been " very religious ". She often used to tell tales, which were rather obviously lies. She was moody and demonstrative, and " always went to full extremes ". She was always tired, and thought a lot about her health. She never took alcohol, and rarely smoked.

History of present illness.—Her present symptoms began 3 months before her admission. She had then been a probationer nurse for one month, and was finding the work too hard. Her only friend among the nurses had just left ; and after much hesitation she had just terminated a rather close friendship with a married man. She then began to feel miserable and tired, had attacks of palpitations and flushing, and would often sigh. She did not notice any dyspnœa on exertion. She missed one menstrual period and the next one lasted 5 weeks, during which she was admitted to the wards because of a fainting attack. She then went off her food, vomited, had dysuria and retention of urine, and began having terrifying dreams. Arrangements were made to give her an anæsthetic for a pelvic examination. Five hours after the operation and shortly after she had been told that nothing abnormal had been found, she had her first fit. She then remained in bed, having frequent fits, day and night (hourly for part of the time), and persistent vomiting. She was then transferred to the Maudsley Hospital.

Condition on admission.—Physically she was of small pyknic build, well nourished, and slightly pallid. Examination revealed only a general increase of reflexes and clamminess of her skin. Blood-pressure 120/80, pulse 80, respirations 20 ; she never showed any pyrexia. Her blood-count showed 12·1 grm.% of hæmoglobin and 4½ million red cells (this responded to iron treatment), but no other abnormality.

She was rather sullen, but with a little persuasion would talk quite freely. An examination of her mental state revealed no abnormality other than intelligence on the lower limits of normal, and hysterical moodiness. The psychogenic factors and the nature of the fits are discussed later. She was having about 16 fits in the day and 2–3 in the night.

The following estimations were found to be within normal limits : blood bromide, 25 mgrm.% ; blood sugar after 14 hours' starvation, 95 mgrm.%. Cerebro-spinal fluid : calcium, 4·9 mgrm.% ; protein, chloride and cells normal. Wassermann reaction negative in blood and cerebro-spinal fluid.

Progress.—After admission she was gradually trained to normal habits of eating, micturition and general behaviour, and came out of her listless state. By the end of 6 weeks she was sufficiently co-operative to discuss her situation freely, and was assisting in some of the ward duties, though still having about 6 fits in the day and 2 in the night. After 5 weeks' amenorrhœa she had a period lasting 5 weeks (which removed any suspicion that her previous menstrual abnormality had been due to an abortion, but diminished her hope that she would ever recover). Psychotherapy consisted in discussing possible solutions of the unpleasant features of her situation, but she failed to co-operate.

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During the last few weeks an unsuccessful attempt was made to reduce the number of her fits by hypnosis, but the occurrence of a fit as soon as the hypnosis became moderately deep would terminate rapport.

Finally, for financial reasons, she had to leave hospital and went as a voluntary patient to a mental hospital. During her stay there her fits soon diminished in frequency to about 1 per week, and she was discharged after 6 weeks. After a further 6 weeks of good health and hardly any fits, she " completely collapsed " and began having frequent fits, and was readmitted to a general hospital.

Description of the Fits.

These occur both at night and during the day. She states that they occur, during the periods of depression with a general hot clammy feeling that come over her, if she cries and " lets herself go ". No other anxiety feature occurs at this stage, during which she knows that she can arrest the onset of the fits by not " letting herself go ". If she does not arrest them, she soon feels a tightness in her chest, followed by tingling in her fingers which spreads all over her and she " goes off ".

Her eyes now turn up, she starts breathing regularly and rapidly (70–80 per minute), and lies limp, with occasional small twitches visible in parts of her muscles, especially in those of her face ; her tendon reflexes are now more exaggerated and Chvostek's sign is positive.

Within half a minute of this onset she begins to toss about violently, often bruising herself, is apparently unconscious, and has the same rapid respiratory rhythm except for interruptions by muscular contortions.

Within a further half-minute she goes off into rigid tonic opisthotonos, with her arms and legs extended, fists clenched and carpopedal spasm. She is now unresponsive to all painful stimuli, but with corneal reflexes present ; the spasm prevents elicitation of Babinski's and Chvostek's signs, and she can be lifted like one complete rod, by a hand placed under the nape of her neck. Her pulse is now slowed (60), full and bounding, her colour is slightly pallid, her skin moist, and her extremities colder and clammy. Her breathing remains the same, with in addition stridor and often foam at the mouth. After about 3 minutes she falls back limp, with her breathing a little easier and slower (60–70 per minute). Then after a further half-minute this sequence of convulsive, tonic and atonic stages is repeated several times till, in one of the atonic phases, her breathing becomes irregular and slower, and within two minutes she becomes fully conscious. She now feels clammy, scared, dyspnœic without palpitations, sleepy, and has a headache which goes off when she is left to go to sleep ; her breathing still remains about 40–50 per minute, but shallow and without apparent distress for a further ½–2 hours.

When she was asked to hyperventilate (40–50 per minute seemed to be the slowest at which she could) she regularly passed into a fit after about 90 seconds : suddenly the respiratory rhythm would jump to its own tempo of about 80 per minute with the other accompaniments following exactly as

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in her spontaneous fits. Not infrequently in the atonic stages of her fits she could be roused by shaking etc., and the fit terminated; but this was not so at other stages. It did not appear that the patient did the overbreathing voluntarily; and she denied this strongly, maintaining that once she gave way to her miserable feelings she could not stop the sequence.

It will be noticed that extreme hyperventilation precedes the motor phenomena of her fits, and is a prominent feature of all the stages, while at the height of the fit she is unconscious. The feeling of tightness in her chest, paræsthesiæ in her extremities, laryngeal stridor and tetanic muscular spasm are present as in hyperventilation tetany, of which she would therefore seem to be rather an extreme form. Generalized tonic convulsions have been described by Geerl Jørgensen (1932) and others as a manifestation in cases of parathyroprival tetany. Other cases of tetany have also been described by Rossett (1924) and others where the position of the hands is not the usual *main d'accoucheur*.

Biochemical Changes During the Fits.

Estimations were made of calcium and titrable alkalinity of venous blood removed before and during the fit (tonic stage) on several occasions. On another occasion samples of arterial and venous blood were withdrawn under oil, at similar stages, for pH and total CO_2 content. Another morning, under starving conditions except for the last sample, urine was collected by catheter for measured periods before, including, and after a fit. All these results are recorded together in Table I, where it will be seen that the only significant change observed during a fit is a slight gaseous alkalosis which is confirmed by all the methods used. The slight rise of blood calcium during the fit may not be beyond the limits of experimental error in these results, but a similar slight but significant change has been found in this type of tetany by Grant and

Table I.—*Biochemical Changes during Fits.*

Venous serum (vacuum venule). (Figures in brackets of a second series of observation.)		Arterial blood (under oil) with anti-coagulant dye.		Venous blood (under oil) with anti-coagulant dye.		Catheter urine.			Specimen times.
Calcium (mgrm. per 100 c.c.).	Titrable alkalinity.	pH.	Total CO_2 (vol.%.)	pH.	Total CO_2 (vol.%).	Vol. per hour.	S.G.	pH.	
10·3 (8·9)	0·0312 N. (0·027 N.)	7·43	57	7·39	68	26 c.c.	1023	5·0	Before fit.
10·4 (9·9)	0·0318 N. (0·029 N.)	7·46	50	7·42	60	190 „	1004	6·0	During tonic stage of fit.
(10·4)	(0·027 N.)	..	..	..	..	190 „	..	4·8	After fit.

Note.—While all the above observations were not taken on the same day, in each case the comparable observations (before, during, and after the fit) were taken on the same day. The series of results recorded in brackets in the first two columns were taken on another day—during a period of alkali therapy, instead of acid.

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Goldman (1920) and several others. The cerebro-spinal fluid was withdrawn on one occasion immediately after the tonic stage of a fit, but the violence of the fits in this patient prevented any proper investigation of the cerebro-spinal fluid changes. (For what it is worth, this specimen removed during the fit showed calcium of 5·4 mgrm. per 100 c.c., compared to 4·9 mgrm. per 100 c.c. of the resting specimen of another day.) McCance (1937) and Carmichael (1937) have shown rather conclusively that any calcium changes in the cerebro-spinal fluid are of little significance in this condition, despite the findings of Barnes and Greaves (1936).

Calcium chloride (8 c.c. of 5%), injected intravenously during the tonic stage of the fit, was found always to arrest the respiratory and motor phenomena of the fit within about 4 seconds, and the patient would be completely awake within 2 minutes of this injection. Practically identical results were obtained by the injection of calcium gluconate (8 c.c. of 10%), but after a slightly longer latent interval (15 seconds) and less suddenly. The application of a rebreather bag and mask would also arrest the fits after a slightly longer

TABLE II.—*Effects of Injection of* 8 *c.c. Calcium Chloride* 5% *Intravenously during a Spontaneous Fit.*

Time.	Respirations per minute.	Observations.
0 minutes	32	Started tossing, and deep breathing.
3–6 ,,	65–75	Tonic stage; stertorous breathing; foaming at mouth.
6–9 ,,	55–65	Tossing.
10 ,,	65–72	Tonic stage recommenced.
12 ,,	72	Still fully-developed tonic stage; injection of calcium given.
5 seconds after injection	..	Lies back quiet and limp with only an occasional shallow respiration.
7 seconds after injection	40	Establishment of shallow regular breathing; still lying, quiet, atonic, apparently asleep.
30 seconds after injection	40	Eyes open and she gradually awakens.
2 minutes after injection	40	Fully awake, talking, lying listless; mentions that she has a headache, but apparently desirous of not being disturbed and she goes off to sleep.
2 minutes till 135 minutes after injection	40–58	Lying lax, limp, sleeping most of the time, with shallow breathing.
140 minutes after injection	25	Ditto.

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TABLE III.—*Effects of Injection of Calcium Gluconate* 10% 8 *c.c. under the same Conditions as those in Table IV.*

Time.	Respirations per minute.	Observations.
15 seconds after injection	40	This was the first sign of any effect, and she now became atonic, with regular shallow breathing.
60 seconds after injection	40	Begins to awake.
2 minutes after injection	..	Fully awake, as at this stage in Table II.

TABLE IV.—*Effects of Application of Rebreather Bag, and of Intravenous Injection of Sterile Water during an Induced Fit.*

Time.	Respirations per minute.	Pulse per minute.	Observations.
0 minutes	40	88	Asked to overbreathe deeply.
1 minute	40	80	Small fibrillary twitchings in muscles.
2 minutes	60	65	Eyes turn up; rapid breathing suddenly sets in; she lies limp. Chvostek +.
2½–3½ ,,	60	60	Tossing about.
3½–6 ,,	70	60	Tonic stage with stridor; foam at mouth. Injection of sterile water at 4 minutes without any observable effect.
6–7 ,,	66	64	Tossing about.
7–9 ,,	70	62	Tonic stage.
9–9½ ,,	66	62	Tossing about.
9½ ,,	70	62	Tonic stage.
10 ,, (bag 0 m.)	70	62	Application of rebreather bag.
10½ minutes (½ m.)	44	64	Became lax, with occasional tossing, and breathing markedly deeper.
12 ,, (2 m.)	36	70	Awake and responded with signs. Bag removed and she talked normally.
13 ,,	40	70	Breathing becoming shallower.
14 ,,	58	86	Breathing very shallow; from now it gradually decreased in rate.
45 ,,	28	80	Asleep.

interval (30 seconds); this did not occur with the mask open to the air. The above observations involving venepuncture were controlled by the injection of sterile water on other occasions, and a considerable pause was always made after the prick before the injection of the fluid. Thus raising either the blood calcium or the alveolar CO_2 tension was sufficient to arrest the fit, which confirmed its tetanic character (Tables II–IV).

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By trial periods at different stages of her treatment, acid or alkali in large doses by mouth, and also calcium both by injection and orally, were found to have no influence on the tendency to spontaneous attacks (Table V). A short period of 4 days on luminal gr. j *t.d.s.*, had only the effect of making her more drowsy. Some observations on the pharmacological responses of this patient to vegetative drugs are recorded in Tables V–VIII; while they are definitely

TABLE V.—*Absence of Effect of Treatment with Acid, Alkali and Calcium on the Frequency and Duration of the Spontaneous Fits.*

Dates.	Treatment.	Number of fits per 24 hours.	Average total duration of fits per 24 hours.
July 29–Aug. 11	*Nil*	11 (9–14)	100 minutes.
Aug. 11–Aug. 22	,,	5 (3–7)	50 ,,
Aug. 22–Aug. 29	Am. chlor. by mouth, 90 gr. per day	5 (3–7)	50 ,, (30–70).
Sept. 5–26	Am. chlor. by mouth, 225 gr. per day. Ca. gluconate intramuscularly, 30 c.c. 10% per day	4 (3–7)	40 minutes.
Sept. 26–30	*Nil*	5 (3–7)	40 ,,
Oct. 1–17	Alkali by mouth (pot. cit. and sod. bic. āā, gr. 240 per day)	5 (4–7)	40 ,, (20–50).

TABLE VI.—*Effects of Injection of Pilocarpine gr.* $\frac{1}{3}$ *Subcutaneously under Starving Conditions.*

Time after injection.	Pulse.	Respirations.	Blood-pressure.	Saliva.	Observations.
0 minutes	70	22	110/80	..	..
4 ,,	84	22	..	(started)	Flushed, feeling hot.
8 ,,	70	22	110/80	..	,, ,,
12 ,,	68	22	125/80	..	..
15 ,,	90	22	125/75	..	Started sweating.
30 ,,	80	22	130/90	250 c.c. (to this time)	Feeling cold, attacks of shivering.
55 ,,	81	54 (deep)	110/85	50 c.c. (further)	Cold clammy extremities, body hot and sweating; still attacks of shivering.
60 ,,	88	52	..	..	Still the same. Jerks ++.
80 ,,	86	50	120/85	75 c.c. (+) (further)	Attacks of shaking and coldness of extremities diminishing.

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TABLE VII.—*Effects of Injection of* 1 *c.c. Adrenaline Subcutaneously under Starving Conditions.*

Minutes after injection.	Pulse.	Respirations.	Blood-pressure.	Observations.
0 minutes	70	30	120/80	..
5 ,,	82	30	120/70	..
10 ,,	80	40	120/65	Palpitations felt.
15 ,,	84	42	130/65	,, ,,
20 ,,	80	35	130/65	,, ,,
30 ,,	80	35	130/65	,, ,,
45 ,,	80	35	130/65	,, ,, but less.
50 ,,	80	35	130/70	No palpitations.

TABLE VIII.—*Effects of Injection Intravenously of Atropine (Danielopoulu).*

Time and injections.	Pulse-rate. Resting.	When standing.	After lying down again.
Before start	70	..	..
Injection gr. $\frac{1}{100}$. 0 minutes.			
5 minutes	80	90	80
13 ,, Injection gr. $\frac{1}{100}$.			
18 ,,	99	110	98
21 ,, Injection gr. $\frac{1}{100}$.	95	..	..
26 ,,	104	105	100
41 ,, Injection gr. $\frac{1}{200}$.			
46 ,,	100	112	100
66 ,, Injection gr. $\frac{1}{100}$.	102	..	..
71 ,,	100	120	100

Results.—P. = 70 (original pulse); S. = 100 (maximum pulse in recumbent position). R.V. (relative inhibitory value of vagus) $= \frac{30 \times 100}{70} = 43$.

abnormal and might indicate some factors in the liability to spontaneous fits, no conclusions have been drawn.

PSYCHOGENESIS.

The patient's family had been unstable, her home life unsatisfactory, and her personality in many respects hysterical from childhood. Definite hysterical symptoms had appeared at the age of 15. The present illness may be attributed to the difficult situation in which she found herself at its onset—with work beyond her capacity, her lover given up, and no friends. Vasomotor and menstrual symptoms, anxiety, and later such hysterical disturbances as vomiting and retention of urine supervened.

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It is of interest that the first fit occurred after hearing that the examination which she had just undergone offered no hope of treatment, while she was only partially recovered from the anæsthetic. This fit might be considered as an emotional outburst similar to crying or a fit of temper, modified by her physical state of partial consciousness, starvation, etc. However the fits were caused in the beginning of the illness, in its later stages they almost ceased only when her environment could not be dominated by them, or it was easy and pleasant, and the mechanism of precipitation at this stage would appear to be essentially hysterical.

She did not respond to psychotherapy, and she was unwilling to follow the suggestion that she might give up nursing (for which she was intellectually and emotionally unfitted). Adaptation in society was also made difficult by her dependence on her mother (for whom she wished to make sacrifices), by her expressed aversion to friendship with men, and by her pride in being "unsexed". These were based on long-standing faulty emotional development and training. She had no desire to change her present status or outlook until her dismissal from her hospital staff, and the associated necessity of being treated in a mental hospital, altered the situation, and gave her an impetus to get better, which psychotherapy alone had been unable to accomplish. The course of events following this change would confirm the view that the condition was mainly psychogenic, using pre-existing mechanisms in the typical hysterical way. As shown by her later relapse, the present state of her mental health is still precarious.

Discussion.

There can be little question that this is a case of spontaneous hyperventilation tetany. The ascertained association of the fits with a slight gaseous alkalosis, due to overbreathing, is in line with the recognized findings in experimental hyperventilation tetany in normal men (Collip and Backus, 1920; Grand and Goldman, 1920, and others), and also with the abnormalities that have been found in most cases of spontaneous hyperventilation tetany. Even in McCance's (1932) patient, in whom he was unable to show any biochemical change, tetany did not develop when the alkalosis was prevented by the use of a rebreather bag during the overbreathing. Apparently one significant abnormality in these spontaneous cases is their excessive liability to tetany from overbreathing and its associated gaseous alkalosis. Rosett's placing of the normal limits for the time of onset of tetanic signs during overbreathing within 10–30 minutes contrasts sharply with the rapidity of their development in these cases—the present patient being, in that respect, perhaps the most extreme reported. Nevertheless the correction of the gaseous alkalosis, or intravenous calcium, removes the tetany, just as it does tetany induced by experimental overbreathing in normal men. The shortness of

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the latent period after the injection of calcium chloride before the arrest of the fit in this patient (5 seconds) suggests a central mechanism for the phenomenon (possibly carotid sinus, possibly cerebral); this explanation of the tetany is also favoured for the more classical cases of hyperventilation tetany by Rosett (1924), Flick and Hansen (1925) and others.

In this patient there were during the illness menstrual abnormalities suggestive of hyperœstrinæmia (or deficiency of the antagonists), which might be correlated with the observations of the increased susceptibility to tetany with hyperœstrinæmia in parathyroidectomized animals (Mathien, 1934), and patients (McCullagh and Kearns, 1935). This endocrine abnormality, and the automatic imbalance shown by the vegetative pharmacological tests in this patient, might be factors in the abnormal susceptibility to tetany in this condition.

The other striking feature in this condition is the spontaneous onset of the attacks. The overbreathing seemed to supervene in this patient when an affective state of irritability and depression developed. Goldman (1922) has recorded attacks of spontaneous hyperventilation tetany during pain, nausea, acute infections and excitement. The attacks in this condition may be compared with anxiety attacks, sighing, crying, vaso-vagal attacks, and the "suffocating turns" described by Baker (1934). Christie (1936), in a study of respiratory tracings in neurotics, has described periodic sighing as characteristic of hysterical patients. In severer cases he found these replaced by short periods of overbreathing, during which tetany sometimes developed. A tracing taken from the patient described here under basal metabolic rate conditions shows these features. Thus, while the physiology of this abnormality of respiration remains obscure, it would seem to be closely related to sighing and crying.

Of interest in this case is the close intermingling of psychogenic factors with physiological abnormalities of a vegetative and biochemical nature. While the liability to tetanoid fits may be constitutional, it is of interest to note their occurrence during whooping-cough in her infancy, due presumably to purely physical factors—anoxæmia, effects of coughing, and possibly cerebral vascular damage. Shannon (1935) regards many of these childhood fits as partly tetanoid in character. To what extent in the present illness physical causes participated with the psychogenic ones discussed it is hard to assess; mild anæmia, starvation, anæsthetic effects, depletion of calcium by menorrhagia, and possibly persistent infantile cerebral damage, etc., may all have had some influence.

Spontaneous hyperventilation tetany is perhaps most frequently found in association with a hysterical illness, though loss of consciousness, as seen in this patient, is rare. Guttmann (1927), in discussing the occurrence of tetany in hysterical patients, considers that it is a preformed mechanism (probably constitutional) apparently released by a combination of anxiety (angst)

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and over-breathing, since in the attacks both of these features are observed to precede the tetany. The predisposing situation to such illnesses is frequently one in which a patient of excitable and hysterical personality finds herself unable to get any enjoyment and " life ", and the illness may be ushered in by a period with much crying and irritability. During the illness signs of anxiety and vegetative instability (flushing, vomiting, etc.) are also frequently seen. The fits seem closely related to so-called affect epilepsy, and are possibly functional disturbances intermediate between the hysterical and epileptic types of fit. At the Maudsley Hospital, there have been several cases complicating a hysterical neurosis, but it has been found that the attacks generally disappeared when the patient was able to make a satisfactory adjustment to her environment. Three illustrations are quoted briefly below :

CASE 1.—Miss B. S—, aged 17. She was admitted because of " heart turns " (epigastric pain, nausea, feeling of suffocation, and giddiness and faintness) of three months' duration. During the latter part of the illness these were associated with tingling and tetanic spasms of hands and legs. She had always been treated as " delicate " by the family who had very strict puritanical standards of conduct, and the patient reluctantly led a restricted life. Physical examination revealed no abnormality other than a congenital heart lesion of mild degree. Blood-calcium 11·3 mgrm. per 100 c.c. Following a discussion of her problems and possible adjustment to them, she was discharged free of attacks and has remained so for 2 years.

CASE 2.—Miss R. D—, aged 28, was admitted complaining of severe loss of weight and of energy, vomiting, headache, irritability, and frequent crying and " turns ". She has always been shy and easily frightened, and had been the " delicate ", spoilt child of the family. After winning a scholarship she spent 7 years at an art school and has since done " free-lance work ". Her family are all very strict Roman Catholics, but since going to the art school the patient has led a rather " wild " life (which finally culminated in an entanglement with a married man). Since attending the art school she has been subject to " turns " of suffocation with tingling and stiffness in her hands and legs when in embarrassing or difficult situations ; and during the year before admission, these have been very frequent, and associated with the other symptoms which confined her to bed most of the time. After 6 months' treatment in the hospital she is (3 months later) now back at interesting work, which does not involve the " artistic " life, and she is free of symptoms.

CASE 3.—Miss E. B—, æt. 23, was admitted for investigation of attacks of spontaneous hyperventilation tetany which she had had about 3 or 4 times a year since the age of 14, her only other symptom being general asthenia. The attacks occurred if she became over-excited (in any way), over-tired, or over-heated (e.g., in hot baths) and were quite typical—first faintness followed shortly by palpitations and a sense of suffocation which made her breathe heavily and culminated in tingling and stiffness of her extremities. During her childhood she lived with an uncle who was a drunkard, and of whom she was terrified. She had always been timid, " delicate ", and easily tired, she has led a quiet life mostly with her mother, and disliked her work. Her condition is unaltered as yet : physical examination revealed no abnormality. Blood-calcium 10·5 mgrm. per 100 c.c.

It is relevant to note the description of hyperventilation tetany in spontaneous hypoglycæmia by Wilder (1934), in encephalitis lethargica by Barker

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and Sprunt (1932), Harrop and Loeb (1923) and others. West (1931) described fits very similar to those in this patient in a child with a parapituitary cyst. The relation of tetany to tonic fits occurring with other intracranial lesions is not clear. It would thus appear that this type of tetany can result from pathological brain lesions or nutritional disturbances, as well as functional nervous disturbances, where it is presumably released by psychogenic mechanisms; but unfortunately, though we know that a gaseous alkalosis is a factor in the manifestations of the attacks, the essential abnormality, shown in the spontaneous onset and the abnormal liability to tetany, remains obscure.

In conclusion, I wish to express my indebtedness for all the biochemical estimations to Dr. Mann and others of the Staff of the Central Pathological Laboratory of the L.C.C. Mental Hospitals. My thanks are also due to Prof. Mapother for permission to publish these results.

Summary.

1. Spontaneous hyperventilation tetanic fits are described in a hysterical patient, who had had apparently similar fits during whooping-cough in infancy. A psychogenesis is shown to be closely related to the present illness. Three other illustrative cases are briefly described.

2. Observations were made of the biochemical changes during the fits, and of methods of inducing and terminating them. A gaseous alkalosis was found to coincide with the fits, and they could be terminated either by correcting this or by giving calcium intravenously; but calcium, acid or alkali therapy had no definite influence on the tendency to attacks.

3. The abnormal susceptibility to hyperventilation tetany, the disturbance of respiratory control seen in these patients and the close intermingling of psychogenesis with physiological abnormalities seen in this patient are discussed; the nature of these latter essential abnormalities is still obscure.

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THE EFFECT OF ALTERING THE CONDITIONS OF THE AUTONOMIC NERVOUS SYSTEM ON THE CHOLINE ESTERASE LEVEL IN HUMAN BLOOD SERUM.

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WITHIN recent years a great deal of attention has been paid to the question of chemical transmission of nervous impulses, and it has been definitely established by Dale and his co-workers (1, 2, 3) that acetyl choline is the transmitter in the case of the parasympathetic nervous system, and also in part of the sympathetic nervous system.

It is impossible to get any direct quantitative measure of the acetyl choline content of human tissues, but the acetyl choline formed in the body is destroyed by a change enzymic in nature. This point has been investigated by Stedman and Stedman (4, 5), and Ammon and his co-workers (6, 7), who have suggested that the enzyme is specific and called it choline esterase.

Using the method of estimating the choline esterase content of human blood sera described in a previous paper (8), a group of thirty normal controls was investigated. The figures obtained showed a range of from 52 to 114 units (μl CO_2 per minute per ml. of serum) at 37° C., with an average of 83 units. Further investigations carried out with a view to establishing any variance determined by food, exercise, sleep, etc., showed remarkably consistent results, the figures invariably remaining constant. Moreover, on keeping the serum several days, no alteration in the choline esterase level was found. As acetyl choline is so intimately concerned with the transmission of nervous impulses in the autonomic nervous system, it seemed desirable to investigate the level of serum esterase in conditions where autonomic imbalance could be demonstrated clinically. A large group of neurotic and psychotic cases, 142 in all, were investigated (see Table I).

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TABLE I.

Diagnosis.	Number of cases.
Anxiety states.	25
Hyperthyroidism	9
Hysteria	6
Manic-depressive psychosis . . .	20
Involutional depression	29
Schizophrenia	13
Senile psychoses	8
Paranoia and paraphrenia . . .	4
Epilepsy	7
Post-encephalitics	2
Others, including endocrinopathies, puerperal insanity, narcolepsy, enuresis, etc. .	19
Total number of cases investigated .	142

Of these cases, 36 were outside the limits obtaining in the so-called normal group; 20 were above the upper limit of normality; of these, 7 were severe anxiety neurotics, 3 were agitated depressives, 3 were manics, 2 were cases of exophthalmic goitre, and the remaining 5 were made up of a vagotonic, an enuretic, a paraphrenic, a hysteric and a case of adrenal cortical hyperplasia. Of the 16 cases with the choline esterase figure below 52 units, 5 were epileptics, 3 were melancholic stupors, 2 were catatonic schizophrenics, 2 were senile depressions, 2 were depressions, 1 was a post-encephalitic, and 1 was a case of Cushing's syndrome with definite evidence of adrenal hyperplasia, e.g. marked hypertension, diminished glucose tolerance, virilism, etc. In this latter case it is of interest to note that the original figure was 104 units, but following two operations, as the result of which the sympathetic nerve supply to the adrenals was cut, the figure dropped to 52 units.

It is quite apparent that these two groups represent entirely different material when considered from the point of view of their autonomic status. In the first group, anxiety with its somatic accompaniments was present in practically every case. In the second group, evidences of autonomic over-activity were entirely absent, and 9 of the cases showed either stupor or a state of dementia where volition was entirely lacking. It was now decided to ascertain what drugs, if any, were effective in altering the choline esterase level in any one case. In this connection, marked inhibition of the choline esterase figure in a group of 12 patients was obtained by using eserine sulphate, gr. $\frac{1}{50}$, subcutaneously. The parasympathetic response following the administration of eserine depends on this inhibition. The figures obtained in this group of cases are shown in Table II (from previous paper, (8)).

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TABLE II.—*Response to Eserine.*

Case.	Age.	Diagnosis.	Choline esterase. Before eserine.	Choline esterase. After eserine.	Variation.
1	62	Paranoia	99	81	−18
2	38	Schizophrenia	78	66	−12
3	48	Agitated depression	81	68	−13
4	56	Involutional melancholia	26	19	−7
5	52	Agitated depression	75	56	−19
6	47	Involutional melancholia	66	56	−10
7	21	Melancholia	70	54	−16
8	18	Melancholic stupor	53	55	+2
9	41	Anxiety state	80	63	−17
10	37	,, ,,	81	68	−13
11	44	Paranoia	54	44	−10
12	55	Disseminate sclerosis	93	78	−15

The limit of experimental error is of the order of 2 units.

TABLE III.—*Response to Prostigmine.*

Case.	Choline esterase. Before drug.	Choline esterase. After drug.	Variation.
1	76	37	−39
2	31	10	−21
3	83	35	−48
4	83	41	−42
5	70	54	−16

Second sample of blood removed 2 hours after 2 c.c. of prostigmine.

With prostigmine, 1 mgrm. subcutaneously, similar results were obtained (see Table III). In this connection it is interesting to note that Stedman (9) has investigated the choline esterase level in the serum of patients suffering from myasthenia gravis and found that the values tend to be low. This excludes any simple explanation of the therapeutic action of prostigmine in myasthenia, and the benefit resulting from the use of prostigmine in this condition does not appear to depend upon the prevention of an excessive destruction of acetyl choline by the esterase. However, the results obtained with eserine and prostigmine simply demonstrate a temporary inhibition of the esterase, which lasts for a few hours, the figure then returning to the previous level. In an attempt to produce a more permanent change in the level of choline esterase in the blood, prolonged stimulation of the parasympathetic nervous system with carbaminoylcholine chloride (Doryl, Merck)

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was tried. This drug has an action similar to acetyl choline, but is destroyed less quickly by the body and its action is, therefore, more prolonged. It was found that the esterase figure was unaltered by a single dose of doryl sufficient to produce somatic indication of parasympathetic activity, such as sweating and frequency of micturition, but was increased by administration of doryl over a period of weeks (see Table IV).

TABLE IV.—*Response to Carbaminoylcholine Chloride.*

Case.	Choline esterase. Before drug.	After drug.	Variation.
1	65	79	+14
2	96	113	+17
3	87	93	+6
4	65	80	+15
5	83	94	+11
6	78	84	+6
7	74	81	+7
8	83	86	+3

Second sample of blood removed after twice daily injections of ·00025 grm. doryl for 10 days.

Clinical experience has shown that bellergal (Sandoz) is beneficial in diminishing the somatic symptoms resulting from autonomic over-activity in neurotic patients. For this reason the effect of prolonged administration of this drug upon the choline esterase activity in a group of patients was investigated (see Table V). One tablet of this drug contains bellafoline ·1 mgrm., ergotamine tartrate ·3 mgrm., phenobarbital 20 mgrm.

TABLE V.—*Response to Bellergal.*

Case.	Choline esterase. Before drug.	After drug.	Variation.
1	76	57	−19
2	91	74	−17
3	111	98	−13
4	78	85	+7
5	87	61	−26

Second sample of blood removed after 1 tablet bellergal (Sandoz) given *t.i.d.* for 10 days.

Several *in vitro* experiments were carried out, using special flasks, which permitted the addition of a substance after the reaction had proceeded for

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sufficient time to calculate an activity value. Using this method, the activity after one half tablet had been added to both reaction and control flasks was approximately one half the activity before the drug was added.

The precise mechanism of this inhibition has not yet been fully elucidated, but the significance is that the decrease in esterase activity shown by the patient to whom bellergal was administered is very much greater than would be expected if the decrease was due entirely to a direct inhibitory action of the drug.

Discussion.

Any attempt to measure cholinergic activity indirectly by assuming that the choline esterase in serum varies as the production of acetyl choline at the nerve-endings is at present premature. However, when it was found, as the result of investigation of the choline esterase figures in a large group of patients, that the cases showing figures considerably higher than those of the apparently normal controls had, in practically every case, evidences of autonomic over-activity, it seemed reasonable to suppose that this autonomic activity was in some way connected with the high esterase content of the blood. Moreover, the cases showing choline esterase levels below those of the normal controls were in marked contrast to the previous group and, as already stated, 9 of the 16 cases were either in a state of stupor or, in the case of the demented patients, showing little or no movement by virtue of a complete lack of volition. Of the remaining 7, 4 were epileptic and 2 were depressives, while 1 had had an adrenal sympathectomy. Thus the evidence presented by the clinical material was in favour of the concept that the level of the choline esterase in the blood is to some degree proportionate to the state of autonomic activity. If the body responded to excessive cholinergic activity by an increase in the choline esterase activity, then this effect ought theoretically to be capable of reproduction by prolonged stimulation of the parasympathetic nervous system by drugs. The evidence from using doryl, although by no means conclusive, supports this contention.

With bellergal a definite fall in the level of choline esterase was obtained, and evidence is adduced to show that this is not wholly attributable to any direct inhibitory action of the drug. It is believed that the clinical improvement found on using this drug in neurotic cases is dependent upon its stabilizing effect upon the autonomic nervous system, and that this factor is partly responsible for the fall in the level of the choline esterase.

Unfortunately, as yet we have not had the opportunity to follow up our case material, and correlate the level of the choline esterase with alteration in the clinical state.

We have attempted in this paper to indicate a possible correlation between the choline esterase level in the blood and the clinical evidences of autonomic

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activity, but fully appreciate that many other factors must be considered in this connection.

We wish to express our thanks to Prof. Henderson for providing us with facilities for this work, and to the Medical Research Council for a personal grant to one of us (H. T.).

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THE UREA CLEARANCE TEST IN PSYCHOTICS.

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IN the routine examination of the urine of patients admitted to this hospital one is struck by the frequency of albuminuria. For example, 27·4% of 667 consecutive female admissions had a trace of albumin in the urine (i.e., a faint cloud appeared when the heat test was applied), while 37·2% had more marked albuminuria (i.e., the urine showed a thick cloud when the heat test was applied); only 35·4% were entirely free from albuminuria. Of 517 consecutive male admissions 20% had a trace of albumin in the urine, 19·5% more marked albuminuria, and 60·5% were free from albuminuria. B. H. Shaw (1) at Stafford found even higher percentages in his 250 admissions in 1935.

Though the factors causing albuminuria are many, such as chronic nephritis, alcoholism, toxic states, febrile states and endocrine dysfunction, these figures were so striking that it seemed worth while to examine the renal efficiency of a group of patients.

Shaw examined the renal function of 44 patients suffering from various types of mental disorder by Rosenberg's modification of Volhard's method. By this method the urine is collected for 24 hours in separate portions under certain standard conditions. The adopted normal criteria are that the specific gravity should reach 1027 in at least one of the specimens, and the total volume in 24 hours should not exceed 750 c.c. He found defective concentration in 34% of the cases examined, and polyuria in many cases, especially in the manic-depressive and melancholic groups.

The chief function of the renal tissue is to eliminate solid substances from the blood in aqueous solution. An important characteristic of the normal kidney is its power of eliminating the required quantity of solids regardless, within wide limits, of the amount of water available for their solution. In most cases of renal disorder, especially in the stage of compensation, examination of the nitrogenous constituents of either urine or blood does not yield any valuable information regarding the degree of functional impairment. The introduction of simultaneous analyses of blood urea and urinary urea by Grehaut in 1904 constituted a great advance in the study of renal efficiency. Then followed Ambard's famous coefficient, now largely given up, but of importance as the first attempt to express mathematically renal functional

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capacity. The blood-urea clearance determination constitutes the most sensitive method at present available for the estimation of the degree of functional renal damage.

Eleven cases of epilepsy and 34 cases of melancholia have been examined by the method of Addis modified by Van Slyke (2, 3). This method is intended to measure the renal efficiency in terms of the number of cubic centimetres of blood cleared of urea by the urine per minute; in other words, the volume of blood which contains the same amount of urea as is excreted per minute in the urine.

Procedure Employed.

After a light breakfast from which tea, coffee and other diuretics are excluded the bladder is emptied and the urine passed is discarded. The urine is then collected for the next two consecutive hours, separately. During the second hour a small sample of blood is taken (0·5 c.c.) from the median basilic vein and oxalated. The following estimations are carried out without delay:

Urine, 1st hour: Volume, urea content.
Urine, 2nd hour: Volume, urea content.
Blood: Urea content.

The method of analysis employed was a micro-technique using the Conway unit and Conway micro-burette (4), which we have found to give highly satisfactory results. It may be briefly described as follows:

The unit is like a little saucer covered with a ground-glass lid and has an inner compartment. 1 c.c. of standard acid (0·00667 N.) is put in the inner compartment, and 0·2 c.c. blood with 1 c.c. water and 0·05 c.c. of a buffer solution (consisting of 6·9% dihydrogen sodium phosphate and 17·9% disodium hydrogen phosphate) are put in the outer compartment, along with one half of a 25-mgrm. tablet of urease Dunning, suitably ground, and the cover placed in position. The reagents in the outer compartment are mixed by gently rocking the unit. This is placed in the 37° C. incubator for 15 minutes. Then 1 c.c. saturated potassium carbonate solution is added to the outer compartment and mixed with the other reagents by rocking the unit. After a further hour's incubation the acid in the inner compartment is titrated against 0·00267 N. sodium hydroxide. The urea plus ammonia content of the urine is estimated by the same method, using 0·2 c.c. of a 1 : 50 dilution of urine. The ammonia content is simultaneously estimated, using 0·2 c.c. of a 1 : 4 dilution without, of course, urease. The urea content is obtained by subtraction.

The volume of blood completely cleared of urea =

$$\frac{\text{Urea content of urine}}{\text{Urea content of blood}} \times \text{volume of urine per minute.}$$

When the volume of urine excreted exceeds 2 c.c. per minute, the clearance

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TABLE I.

No.	Initials, sex, age.	Duration of mental illness.	Physical condition at time of test.	Mental condition at time of test.	Albuminuria at time of test.	Blood urea (mgrm. %).	Urinary urea (grm. %).	Urine volume c.c. per hour.	c.c. blood cleared per minute.	Renal efficiency (%.)
	C. E. H—, female, 53 years	2 years	General debility	Depressed, delusional, hallucinations of hearing and sight, deterioration of personality	Faint trace	40·2	3·29 3·88	27 33	55 62	102 116
2	M. B. R—, female, 50 years	2 ,,	,, ,,	Depressed, delusional, auditory and visual hallucinations, deterioration of personality	,, ,,	20·8	2·00 1·95	37 38	74 75	137 139
3	A. M. W—, female, 43 years	2 ,, recurrent since aged 28 years	,, ,,	Chronic melancholia with deterioration of personality	,, ,,	22·2	1·79 1·46	130 102	175 86	230 158
4	A. J—, female, 52 years	2 years	Fair	Melancholia, visceral delusions, ideas of unworthiness, stereotyped attitude, resistive	Trace	19·4	2·45 3·03	29 31	88 112	163 207
5	E. D. H—, female, 42 years	1½ years	Underweight	Melancholia, perplexed state, retarded thought and action	—	17·0	2·13 2·04	17 23	67 74	124 138
6	A. M—, female, 51 years	1 year	Fair, well nourished	Melancholia, mildly agitated, retarded thought and action, ideas of ruin	—	15·4	2·04 2·34	20 31	76 109	141 202
7	H. M. C—, female, 56 years	1½ years	Undernourished	Involutional melancholia, agitated, apprehensive, difficult with food	Trace	22·6	1·43 1·15	19 57	36 50	67 93
8	M. G—, female, 52 years	1 year	Fair	Melancholia, ideas of contamination, now convalescent	—	32·2	2·37 2·75	34 36	55 66	102 122
9	A. W—, female, 55 years	2 months; recurrent since aged 50 years	,,	Melancholia, retarded, lack of confidence	—	13·8	0·64 0·77	75 70	52 60	96 111
10	N. F—, female, 60 years	18 years	General debility	Chronic melancholia, stereotypies of speech and gait, delusions of impending burning	—	32·2	2·78 3·23	27 21	58 59	107 109
11	A. G—, female, 43 years	3 years	Fair	Depressed, delusions of persecution, hallucinations of hearing, antagonistic, abusive	Trace	21·0	2·82 2·34	11 17	58 59	107 109

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TABLE I—*continued.*

No.	Initials, sex, age.	Duration of mental illness.	Physical condition at time of test.	Mental condition at time of test.	Albuminuria at time of test.	Blood urea (mgrm. %).	Urinary urea (grm. %).	Urine volume c.c. per hour.	c.c. blood cleared per minute.	Renal efficiency (%).
12	B. L—, female, 65 years	1 year	Fair	Convalescent from attack of melancholia	Trace	20·2	1·94 2·01	32 40	70 81	130 150
13	D. M—, female, 54 years	2½ years	General debility	Slowly recovering from profound depression, retarded thought and action	—	25·4	2·42 2·19	30 27	67 58	124 107
14	E. M—, female, 48 years	8 months	Fair	Depressed, retarded, delusions of unworthiness and of causing disease	—	23·8	1·71 1·57	33 43	53 56	98 104
15	J. S. R—, female, 58 years	1 year	General debility	Depressed, agitated, apprehensive, self-absorbed	—	13·0	1·77	33	101	187
16	M. J—, female, 60 years	5 years	Fair	Insomnia, depressed, apprehensive	Trace	20·2	2·04 1·50	21 35	60 50	111 93
17	A. P. M—, female, 60 years	2 ,, recurrent since aged 25 years	General debility	Recovering slowly from long period of depression, lassitude, inactivity	—	25·0	2·79 2·38	19 25	63 61	117 113
18	A. C—, female, 48 years	4 months; recurrent since aged 26 years	Menorrhagia and anæmia	Manic-depressive psychosis; now mildly depressed and retarded	+	16·6	1·70 1·30	21 41	61 65	112 120
19	A. McM—, female, 63 years	4 months; recurrent since aged 33 years	Malnutrition	Agitated melancholia, ideas of sin and impending judgment, hallucinations of hearing, ideas of reference	Faint trace	25·4	1·85 1·66	26 53	48 61	89 113
20	E. B—, female, 48 years	1½ years	Fair	Convalescent from attack of recurrent melancholia with suicidal impulse	—	20·2	1·94 1·47	34 37	72 57	133 106
21	E. E. R—, female, 41 years	1 year	Emaciation	Melancholia, ideas of visceral disease, refusal of food owing to delusions	—	33·8	3·54 2·67	12 17	47 42	87 78
22	I. C. F—, female, 45 years	5 years	Fair	Melancholia with suicidal impulses affective loss; now convalescent	—	30·2	2·48 2·80	8 20	30 54	56 100

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TABLE I—*continued.*

No.	Initials, sex, age.	Duration of mental illness.	Physical condition at time of test.	Mental condition at time of test.	Albuminuria at time of test.	Blood urea (mgrm. %).	Urinary urea (grm. %).	Urine volume c.c. per hour.	c.c. blood cleared per minute.	Renal efficiency (%).
23	C. E. M—, female, 64 years	1 year; recurrent since aged 51 years	General debility	Profound depression, ideas of unworthiness and impending disaster, desire for death, stereotyped attitude, silent and resistive	Faint trace	34·6	1·13 1·55	9 15	13 22	24 41
24	W. G. L—, male, 66 years	2½ years	Underweight	Melancholia, hypochondriacal delusions, deterioration of personality	,, ,,	32·8	1·08 2·38	33 16	24 37	45 68
25	G. S. T—, male, 58 years	5 ,,	Well-nourished	Chronic melancholia, slight retardation, ideas of unworthiness and physical weakness, mannerisms of gait	—	20·8	1·82 1·54	80 72	101 81	187 150
26	J. F—, male, 65 years	10 ,,	Fair	Chronic melancholia, slight agitation, ideas of unreality	Faint trace	32·6	2·24 2·65	118 137	135 185	250 247
27	G. A. S—, male, 68 years	6 months	,,	Melancholia, ideas of unworthiness and ruin, ideas of reference, egocentric, retarded	—	27·4	1·76 1·67	38 40	51 50	95 93
28	A. F—, male, 68 years	2 years	Underweight	Agitated melancholia, fear of impending disaster, ideas of physical weakness, stereotypy of utterance	—	28·0	1·85 1·98	79 47	76 63	141 117
29	D. A—, male, 65 years	2 ,,	Fair	Melancholia, ideas of ruin, lack of confidence, stereotyped utterance	—	20·0	1·59	79	87	162
30	P. G—, male, 44 years	2½ ,,	General debility, flabby, headaches	Melancholia, inertia, vague fears	—	27·0	2·16	63	82	152
31	A. P—, male, 66 years	3½ ,,	Mild diabetes	Agitated melancholia, ideas of reference, suspicious and indecisive	Trace	23·4	1·28 1·59	70 61	59 54	109 100
32	T. B—, male, 45 years	1 year	Renal glycosuria	Melancholia, ideas of unworthiness, despair, self-absorbed	—	20·4	1·63 1·95	177 84	163 78	220 144
33	R. C. H—, male, 51 years	18 years	Fair	Chronic melancholia, mild enfeeblement	—	33·8	2·38 2·46	26 34	46 55	85 102

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TABLE I—*continued.*

No.	Initials, sex, age.	Duration of mental illness.	Physical condition at time of test.	Mental condition at time of test.	Albuminuria at time of test.	Blood urea (mgrm. %).	Urinary urea (grm. %).	Urine volume c.c. per hour.	c.c. blood cleared per minute.	Renal efficiency (%).
34	H. D—, male, 62 years	5 years	Fair	Melancholia, self-absorbed, hypochondriacal ideas	—	27·0	2·14 2·89	16 30	41 76	76 141
35	M. G—, female, 43 years	40 ,,	Epilepsy	Epileptic amentia	+	11·8	1·59 1·27	26 53	89 101	165 187
36	C. H—, female, 38 years	27 ,,	,,	,, dementia	—	17·0	0·82 0·79	76 96	54 59	100 109
37	I. M. L—, female, 54 years	51 ,,	Epilepsy, vasomotor disorders	,, ,,	—	23·0	1·24 0·83	55 74	52 40	96 74
38	C. G. J— female, 35 years	12 ,,	Epilepsy	,, psychosis	+ ; hyaline casts	25·0	1·24 1·80	22 39	30 58	55 107
39	R. H—, male, 28 years	13 ,,	,,	Epileptic psychosis with profound mental enfeeblement	—	20·6	1·37 1·15	68 80	70 64	131 119
40	J. L—, male, 39 years	23 ,,	Epilepsy, varicose veins, rather corpulent	Epileptic personality with minor degree of mental enfeeblement	—	21·8	1·62 1·51	65 49	77 62	141 115
41	F. K—, male, 28 years	15 ,,	Epilepsy (severe)	Epileptic psychosis with mental enfeeblement	—	28·6	2·32 2·87	37 55	61 96	113 178
42	R. O. D—, male, 46 years	21 ,,	Epilepsy (following head injury)	Epileptic personality	—	23·0	2·23 2·92	68 51	103 117	191 217
43	F. W. H—, male, 40 years	28 ,,	Fair, epilepsy	,, dementia	—	19·0	0·79 0·90	57 60	40 47	74 87
44	A. P. P—, male, 44 years	32 ,,	,, ,,	,, personality	—	21·8	1·15 1·38	57 63	51 65	94 120
45	H. J. T—, male, 42 years	25 ,,	Epilepsy (scarlatinal nephritis, age 13 years)	,, dementia	Faint trace	18·6	0·96 0·63	63 77	53 38	98 70

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amounts to about 75 c.c. blood per minute. With urine volumes below 2 c.c. per minute the following formula is used:

$$\frac{\text{Urea content of urine}}{\text{Urea content of blood}} \times \frac{2}{\text{Volume of urine per minute}},$$

for which the standard is about 54 c.c. blood per minute. Using these figures as standard, the results found were expressed as percentages of the standard. The range normally allowable is from 70% upwards. In interpretation it is also to be remembered that if the urinary urea exceeds 2·5% in any specimen it is improbable that renal damage exists, as such a concentration is unattainable by diseased kidneys.

Results.

The table of results shows two figures for almost every patient, the first being calculated on the excretion during the first hour, and the second on the excretion during the second hour.

Figures for the renal efficiency fell below the standards adopted for normality in only 2 of the 34 melancholic patients and in 1 of the 11 epileptic patients studied.

In 17 of the cases which had a slight albuminuria at the time of the test, 6 had a renal efficiency above 130%.

The results indicate that albuminuria occurring in patients suffering from melancholia or epilepsy is rarely associated with renal deficiency.

The two melancholic cases which showed marked renal impairment have certain features in common, and have a bad prognosis. The following is a more detailed account of these cases.

No. 23. C. M—, female, aged 64. Before breakdown this patient was a bright attractive person who always had plenty of friends. There were no previous mental illnesses. In 1924, when she was 51 years old, she had a vague abdominal condition which was diagnosed as "enteroptosis", and while complaining of this pain, she quite suddenly became depressed and suicidal. She recovered after a few months' institutional treatment, and had a remission which lasted seven years. She broke down again in 1931, and since then has had four short remissions. Her condition now is one of profound depression with the most devastating ideas of unworthinesss and auditory hallucinations accusing her of evil practices. She is very retarded and answers in short sentences with difficulty. She says she is the cause of untold suffering to others, and that she is about to be burned. She sits in a stereotyped attitude, is resistive and difficult with food, and sleeps badly. She has been in this depressive state for over a year, and the outlook is very unfavourable. Physically she has lost much weight; the urine contains a trace of albumin, and occasionally a few hyaline casts.

No. 24. G. W. L—, male, aged 66. This patient before his breakdown was a vigorous responsible man of well-balanced mental type. His mental illness seemed to follow directly on a physical illness. In December, 1933, he suffered from erysipelas of the groin, followed by septicæmia, and then by low-grade infective arthritis of hips and spine. Almost immediately after the septicæmia he showed signs of depression with strong suicidal impulses. Since then he has improved a little at times, but always relapsed again. His condition is one of profound

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depression with ideas of physical disease and persistent pessimism. He sits in a stereotyped posture, resisting efforts to help him, soils himself occasionally, and has filthy habits. He swears and is abusive when coaxed with food, and when alone sits gibbering his perseverations of physical ruin and hopelessness. He is troubled with insomnia. Physically his condition is poor; he is very much underweight, and his urine contains albumin and occasionally hyaline casts. We consider the prognosis very bad.

I have to thank my assistant, Miss E. Semeonoff, B.Sc., for much technical assistance and valuable advice, and Dr. C. C. Easterbrook, Physician Superintendent, for permission to publish the cases.

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A NOTE ON THE USE OF DIATHERMY IN IDIOPATHIC EPILEPSY.

By J. L. CLEGG, M.D., D.P.H., D.P.M.,
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IN 1935 V. V. Issahyev reported very favourable results from the treatment of a number of cases of idiopathic epilepsy by diathermy to the head, stating that although the method could not be held to cure the complaint, nevertheless it produced considerable relief in the majority of cases. The object of the present investigation has been to repeat this form of treatment in a further series of cases in order to see how far these results could be confirmed.

LITERATURE.

Issahyev treated 24 cases, 12 of each sex, whose ages and duration of affliction varied within wide limits. He specially selected patients in whom the fits were frequent, in order to obtain quick results, and included both *grand mal* and *petit mal* in his series. The treatment was given on alternate days and the number of applications varied from eight to twenty, the course being repeated at four-monthly intervals in three of the severest cases. No other treatment was given and the patients continued to carry out their usual daily routine.

After one year's observation he was able to report improvement in 75% of the cases, and deterioration in none. He found that the frequency of attacks was diminished for more or less protracted periods, occasionally up to one year; the individual attacks lessened in intensity and the general condition of the patient improved. Endocrine disturbances decreased the efficiency of the treatment, and in these cases it was found necessary to use opotherapy in addition. The effectiveness of the cure did not show any relationship to the duration of the illness, but those having the largest number of fits did the best.

TECHNIQUE.

In the present series 11 cases were treated, all males, patients of the South Yorkshire Mental Hospital. In every instance the fits were frequent, averaging at least ten per month, and in no case was there an endocrine basis. No alteration was made in any treatment which had been employed up to the time of starting diathermy, such as the administration of luminal, nor any change made in the ordinary routine of institutional life.

The patient was placed in a reclining position and a suitably covered electrode, 6 by 12 cm. in size, applied to the neck, which had been adequately shaved. Two similar but slightly smaller electrodes were connected to the other terminal, of which one was applied to the forehead and the other to the epigastrium. All electrodes were moistened with saline, and were kept firmly in position by means of suitably arranged straps. The commencing current

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used was 400 m.a., increased to 600 m.a. at the second sitting, and to 800 m.a. at the third, the latter strength being maintained for the rest of the sittings, which were fifteen in all, each of twenty minutes' duration and given on alternate days.

Results.

Only two of the cases showed improvement, the remainder showing no change. In no instance was the complaint aggravated. In both of the two cases showing improvement the treatment was begun when the patients were in *status epilepticus*, and the improvement showed itself not so much in the shortening of the status as in a modification of the fits in the period succeeding it. In the first case these were greatly reduced in frequency for 4 months, as compared with their incidence before. In the second case there was no difference between the frequency before and after the status, but in the latter period there was a diminution in the severity of individual fits, which were of shorter duration and not succeeded by the long period of confusion which had previously characterized them, and also the patient was improved mentally, and more easily managed. This has lasted up to the present—that is, for ten months.

In view of the large proportion of failures—82%—the investigation was hardly worth continuing as it occupied the time of the staff for a considerable period, and was not likely to become a routine method of treatment for epilepsy. It is not possible to exclude the possibility that the apparent improvement in the two cases described above may have been the quiescent period which often follows a series of fits—a "quiet after storm"—although cases having a large number of fits were those which Issahyev found to do best, and in this respect the present results coincide with his. However, he does not make it clear whether his cases were those in which the frequency had been maintained for a long time prior to diathermy, or whether the fits just happened to be more frequent at the time when the treatment was begun. If the records of epileptics, covering a number of years, are examined, wide variations in the monthly incidence of fits will be found, and it is not justifiable to claim that any form of treatment is successful unless the incidence is permanently reduced in a large proportion of cases.

Summary.

(1) The treatment of idiopathic epilepsy by diathermy has been tried in 11 cases.

(2) With the possible exception of two cases the results were disappointing, and it was not considered worth while to continue the investigation.

I am indebted to Dr. A. Pool, Medical Superintendent of the South Yorkshire Mental Hospital, for permission to carry out this work and publish the results, and to Mr. A. Booth for his care in carrying out the actual treatment.

Reference.—Issahyev, V. V., *Klinitcheskaya Meditsina*, 1935, pp. 1186-90.

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Part II.—Reviews.

A Study of Hypoglycæmic Shock Treatment in Schizophrenia. By ISABEL G. H. WILSON. London: H.M. Stationery Office, 1936. Pp. 74. Price 1s. 3d.

In 1930 Dr. Manfred Sakel, of Vienna, found that deprivation symptoms in the treatment of morphia addicts do not occur if limited doses of insulin are given. In the experimental stages of this therapy states of hypoglycæmia now and then occurred, and the psychological response of certain patients to such states suggested to Dr. Sakel that hypoglycæmia was likely to prove beneficial in actual psychoses. Having enlisted the support of Prof. Poetzl, who is the successor to Wagner-Jauregg as head of the Vienna University Psychiatric Clinic, he began there the hypoglycæmic treatment of schizophrenics late in 1933. This form of therapy has since spread to most continental countries, particularly Switzerland, as well as to Japan and U.S.A.

Attention having been drawn to this in the 1935 report of the Scottish Board of Control, and the method having been introduced at Edinburgh, Dr. Isabel Wilson was instructed by the English Board to investigate the therapy in Vienna and at Münsingen, Switzerland. During her stay she was able to observe all the aspects of the treatment in 30 patients, and to examine 27 patients discharged after treatment. She also had opportunities of gathering the opinions of authorities in Vienna and elsewhere, some of which were favourable, and some critical. Dr. Wilson's report is far more than a review; it sums up admirably the whole controversial matter, and is a mine of information as well as a manual of actual treatment. It contains chapters on technique, clinical observations, theories, results, etc., together with several tables and a complete bibliography.

As is now generally known, the method consists of repeatedly producing, by means of one daily intramuscular injection of a graduated quantity of insulin, a state of hypoglycæmic coma. Beginning with 15–20 units, the dose is increased daily until the dose producing coma is reached. Once the coma dose has been reached, it is repeated daily (Sunday excepted), so long as active treatment is necessary, the usual period being from 4 to 6 weeks. The average coma dose is 80–100 units. The dose is then much reduced (stabilization phase), and eventually stopped.

An intragluteal injection is given to the fasting patient at about 8 a.m. The first hypoglycæmic symptoms set in from 1–1½ hours afterwards. The patient's general condition remaining satisfactory, hypoglycæmia is terminated when it is considered on therapeutic grounds to have lasted long enough (on the average about 4 hours after the injection), by giving a glucose solution by the nasal tube. In emergencies an intravenous injection of glucose is given, which must be followed by the full glucose feed. Epileptiform attacks indicate that hypoglycæmia should be terminated at once. This is effected by an intramuscular injection of adrenaline, which is followed by intravenous glucose and

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the glucose feed by mouth. Treatment is over by 1 p.m. While under treatment the patient requires to be under constant observation of a doctor trained at one of the centres, and of selected nurses, and everything needed for terminating hypoglycæmia must be close at hand. With such precautions treatment does not appear to be unduly dangerous, though it is not entirely possible to avoid risk to life.

Three deaths occurred during the treatment of the first 200 cases in Vienna, but none occurred in Switzerland. The first death occurred in the experimental stage, when the necessity for supplementing the intravenous glucose by the feed was not realized. The second death was due to coronary sclerosis, and it is now recommended that every patient should have an electrocardiogram done before treatment. Electrocardiograph investigations during treatment have shown alterations of the T-wave; these have disappeared within a few hours. The third death was due to pancreatic necrosis; this is a possible risk, although similar animal experiments have shown no changes in the pancreas. Practically all patients, however, gained weight and improved physically, and in the opinion of independent observers did not suffer in any respect from the treatment.

As insulin begins to act, the psychological effects take place in the following order: quietness, somnolence, sleep, coma. The behaviour of individual patients, however, varies greatly. Many show great restlessness and excitement before reaching coma, while others go into coma perfectly quietly. The physical effects appear more or less in the following order, and here again wide individual variations exist: A sebaceous exudate on the forehead, soon followed by copious sweating. The pulse-rate, generally speaking, tends to increase. Muscular movements are very common. They begin with purposive movements of the hands and arms; then, suddenly, psychomotor excitement may set in; eventually the patient lies practically motionless in coma. Finally, movements of an entirely different type may set in: isolated twitching and jerking, beginning in the forearm and spreading to the head and body. These movements may, though not necessarily, merge into generalized epileptiform convulsions.

About 5 minutes after the glucose feed the patient shows the first signs of waking, but he is not fully awake for another 10–15 minutes. During this period the patient manifests the earlier psychological reactions, but in the reverse order. Dr. Sakel speaks of the cerebral functions gradually disappearing, level by level, as the hypoglycæmia deepens, and their rapid return during waking. As the patient awakes, there may be a short phase of psychomotor excitement, followed by one of confusion. Before speech, which at first is disarticulate and paraphasic, returns, the patient understands questions and replies by nodding or shaking the head. Later, patients may resemble persons under the influence of alcohol; they talk in an unconcerned and playful manner for a short while, after which, in those responding, thoughts become ordered. Most patients have amnesia for practically the total hypoglycæmic period until full waking, and in certain cases of prolonged coma there is retrograde amnesia. These hypoglycæmic effects on the higher nervous functions and the various neurological phenomena of hypoglycæmia invite further study and research.

The most striking effect of the therapy is the occurrence of a period of lucidity after injection. Lucidity "does not by any means always occur, but when it does . . . it makes on the observer and sometimes on the patient a very strong impression that something in the nature of a direct

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attack on the basis of the psychosis is going on". Dr. Wilson quotes, in the patient's own words, a strikingly clear and reasonable account of such feeling experienced, and goes on to say that if insulin can in certain circumstances produce such clearness, its possibilities should be explored. At the beginning of treatment lucidity usually does not last longer than the hypoglycæmic period, and for the rest of the day the patient presents his usual appearance. As treatment goes on, however, in a case responding satisfactorily, the lucid periods gradually become longer, extending well into the afternoon, until finally the normal periods have spread over the whole day and the patient is free of symptoms. Temporary lucidity is not difficult to obtain, even in quite chronic cases; the difficulty, however, is to make such periods coalesce, and even a promising start does not necessarily lead to success.

In the beginning of the stabilization phase, what Dr. Sakel describes as "reversed action" of insulin or "reactivation" of the psychosis may occur. That is to say, the patient now shows no signs of his psychosis except during the hypoglycæmic period. It appears that hypoglycæmia, which, in Dr. Sakel's words, inhibits that which is functioning (ordinarily the psychosis) and activates that which is latent, now brings to light what has remained of the psychosis. This is looked upon as favourable, hence the stabilization phase. It is continued until the activated symptoms have gradually subsided, when the patient presents the same appearance within hypoglycæmia as without. Dr. Wilson's own impression is, however, that such reversed action is more apparent than real.

Equally remarkable is, in successful cases, the friendly spirit among patients and the insight they have after treatment. Dr. Müller, of Münsingen, is quoted: "The contact we have with patients treated with insulin is quite a different thing from that with spontaneous remissions. The patients are much freer, more natural, more spontaneous; they make friends with each other when they come to visit and write cards and letters to the doctors. Their outlook towards their illness is a natural one and they can often talk it over without harm."

In a further chapter the rationale is discussed, about which little is known. Clinical observation and experience have been considered of primary importance, and the theoretical foundations may be discovered later, as happened in malaria therapy. The lethargy and sleep produced is not the therapeutic agent, nor the additional attention devoted to the patient, so far as controls can be relied upon. A case is quoted to show that non-specific shock is not the basis of the method, neither is it emotional shock. The insulin effect appears to be sympatheticotonic rather than vagotonic. Investigations into hypoglycæmia and the endocrine balance are being carried out in Switzerland. As quite a number of patients recover without reaching the shock stage, it seems evident that hypoglycæmia rather than shock proper is the therapeutic agent, and Dr. Wilson aptly suggests that "high-dosage insulin treatment" would be a more appropriate title. The effects of large doses of insulin cannot be solely due to the lowering of the blood-sugar. Contrary to expectation, the depth of coma does not necessarily coincide with the lowest blood-sugar value, and coma has occurred with the blood-sugar curve actually rising.

The published results are not easy to interpret, statistics over a sufficiently large number of cases not being available at the time when the report was published. Another difficulty is that the duration of illness plays an important role, those cases with a short history (not over 1½ years' duration) responding very much better than those of longer duration. While agreeing that alleged

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results in these recent cases must be assessed with special care, because of their tendency to spontaneous remission, this should not, in Dr. Wilson's opinion, be allowed to weigh too heavily against the claims made for the method; after all, the chronic schizophrenics constituting the bulk of the mental hospital populations have at some time all been early cases. The results then published from Vienna and Switzerland, being the first 101 and 70 concluded cases respectively, are reviewed in detail, and a small number of cases from other sources. While stating from her own observation that the association between insulin treatment and clinical improvement is occasionally very striking, Dr. Wilson wisely regards the published statistics as tentative only. The Swiss method of assessment, by which no undue importance is attached to the percentage of cases "improved", is to be recommended. They prefer to distinguish between cases of complete remission (i.e., discharged free from symptoms, with insight, and at work in their former occupations) and unchanged cases, by which they signify no improvement, or temporary improve ment only. Looking at the results from Vienna and Switzerland arranged accordingly. complete remission was obtained in 68–80% of cases of not over ½ year's duration, while 13–21% remained unchanged. The chance of complete remission diminishes progressively the longer the illness has lasted before treatment. According to a Swiss table included in the report, the figures for complete remission are as follows: Duration not over ½ year, 80%; from ½–1½ years, 60%: over 1½ years, 4% only. Conversely, cases remained unchanged: duration not over ½ year, 13%; from ½–1½ years, 15%; over 1½ years, 42%. It is not possible to arrange the Vienna figures similarly, as two groups only were recorded—under ½ year's duration and over—but their trend is the same. No relapses occurred in Switzerland, where the period reviewed is 9 months, and 15 occurred in Vienna, where it is 2 years. Several of the relapses remitted or improved after a second course of treatment; in the appended table, however, all relapses, regardless of the subsequent outcome, have been recorded under the heading "unchanged". Such grouping makes the Vienna figures appear less favourable than the Swiss, but such possible understatement is to be preferred.

Taking together the cases regardless of their duration, complete remission was achieved at both centres in 45–50% of the cases, and if to these are added the improved (most of whom were able to return home and work), positive results have been obtained in about two-thirds of the cases. This compares favourably with figures for spontaneous remission, which Dr. Wilson quotes as "all researches taken together—20–30% at most. The acute cases rise over 50%, the old ones are under 10%". As regards the permanence of results, all that can be said is that recoveries which took place in autumn, 1933, were maintained, although this is a relatively short period in the light of Kraepelin's statement that in his time "12·6% of the improvements bore the stamp of complete recovery, which, however, seldom lasted longer than 3–6 years".

Dr. Wilson concludes that the method merits ample clinical trial, and advises that this should be begun in a single public mental hospital, rather than in several simultaneously. The doctor in charge would do well to visit both Vienna and Switzerland to see the method at first hand. Later on, doctors of other hospitals could learn the method from him. Treatment should not be initiated by those who have only read of the method. The arrangement of an insulin ward, apart from other patients, is recommended, and until further experience is gained, it would be wise to limit the number of cases to 6 or 8. One doctor should be responsible for the treatment, and should have

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a guiding hand in all that is done to his patients. He must remain in the insulin ward until the last patient is out of hypoglycæmia. At about mid-day, the approximate time for terminating hypoglycæmia, it is a great advantage to have other doctors within call. Experience is just as important for the nursing staff as for the doctors. The nurses should be carefully selected, and taught by written, verbal and practical instructions. Treatment should be carried out confidently, as it would be a pity to miss the chance of promoting improvement by allowing patients or those in charge to become disheartened. Full records should be kept, and contact maintained with discharged patients for at least five years, preferably ten. H. PULLAR STRECKER.

An Enquiry into Prognosis in the Neurosis. By T. A. Ross, M.D., F.R.C.P. Cambridge University Press, 1936. Price 10s. 6*d*.

This is an admirable book. It has three outstanding virtues: (1) It is factual and gives figures, showing, not what is presumed to have happened, but what actually has happened to a large number of cases which were treated at the Cassel Hospital; (2) it displays an excellent combination of common sense with an adequate understanding of psychopathology—an unusual combination; (3) it reveals Dr. Ross's nationality through his style, which is terse and to the point.

The tables of results are interesting and full—too full to be discussed at length here. The total results are 45% well, 25% improved after 1 year, a figure falling to 34% and 6% after 5 years. The figures correspond roughly to the few sets of figures published. They certainly should give grounds for satisfaction, seeing the severity of the cases treated.

Dr. Ross goes into the pros and cons of hospital versus out-patient treatment, and briefly discusses various types of treatment. He favours analytical psychotherapy, in which analyses of various depth (though never so intense as the Freudian psycho-analysis) are combined with a certain degree of persuasion. He emphasizes the importance of the "moral influence" of the therapist in the Weir-Mitchell treatment, and points out how this, the most important adjuvant, is forgotten by many writers in describing this form of treatment. Hypnotism he does not care for, as its cures are largely symptomatic, and he draws a comparison to the happy, immediate, but nevertheless undesirable results of alcohol and morphia in relieving neuroses.

This is an altogether valuable book, both for the psychotherapist and the practitioner who wants guidance as to what the outcome of neuroses is likely to be. W. L. NEUSTATTER.

Inhibitions, Symptoms and Anxiety. International Psycho-analytic Library, No. 28. By SIGMUND FREUD. London: The Hogarth Press and the Institute of Psycho-analysis, 1936. Pp. 179. Price 6s.

Ernest Jones has said that anxiety is the most important single symptom in psychopathology. Prior to Freud, psychiatry had hardly approached the subject, and any knowledge we now possess upon it is to a large extent due to his pioneer work. It was Freud who first isolated and stressed the importance of the anxiety syndrome. Again it is due to his teaching that we have learnt

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to regard all psychopathic behaviour as an attempt to avoid the arousing of anxiety. In this present work, his many previous contributions to the study of anxiety are shaped into a harmonious whole. This has necessitated certain modifications of previous theories, but nothing vital has been lost.

However satisfying this work may be psychologically, in actual fact it adds very little that is of direct clinical significance ; this despite the statement on the wrapper that *Inhibitions, Symptoms and Anxiety* is the author's most important contribution to the purely clinical side of psycho-analysis during the period since the war. What is learnt here is incontrovertible, once the initial difficulty of conceding to the psycho-analytic concept that all behaviour is derived from constructive and destructive impulses has been surmounted. But however psychologically sound it may be, it is of little help in the elucidation of an anxiety state to realize that the condition is ultimately due to deprivation and non-gratification, modelled on the experience of birth. Contrast these broad concepts with the clinically valuable, if much more disputable, theories expounded in his early papers on, for instance, the anxiety neurosis.

In these Freud taught that in such conditions the causal factor was always to be found in some disturbance of the *vita sexualis*. This theory met with great opposition, and many cases have been published in which the anxiety state has been explained satisfactorily along other lines. On the other hand, practically every subsequent authority has admitted that some cases of anxiety neurosis do support the Freudian theory, and no psychotherapist would attempt now to treat this disorder without first investigating thoroughly the sex life. The same argument holds good for Freud's investigations into the psycho-neuroses. These early papers may have had the fault that they generalized from the particular ; the theories founded upon direct clinical experience may have to be revised. Their importance lies in the fact that Freud drew general attention to the presence of certain ætiological factors in the cases he had studied—factors which had previously been overlooked.

The present work is devoted to broad psychological concepts, schematization and ultimate conclusions. Psychologically profound and extremely interesting, it has only a remote relation to clinical psychopathology. It is written with Freud's usual lucidity and the translation is well done. Contemporary with the present volume, an American translation by M. A. Bunker has been published serially in the *Psycho-analytic Quarterly*. Abstracts of the letter have appeared in the Epitome Section of this Journal, to which the reader is referred for a summary of conclusions. STANLEY M. COLEMAN.

Friendship-Love in Adolescence. By N. M. JOVETZ-TERESHCHENKO. London : George Allen & Unwin, Ltd., 1936. Pp. 367. Price 16*s*.

It is the aim of this book to prove that in adolescence there is a specific mental phenomenon—friendship-love, or more shortly love—which is quite separate from, and is in conflict with, the sexual urge. The essential data upon which the investigation is based consist of the intimate diary of a Russian boy, John Z—, written between the ages 13 years 4 months and 16 years 1½ months. This is supplemented to some extent by the adult John's recollections, and also from other sources. In a short chapter additional matter is brought forward, extracts from diaries, letters, etc., of other adolescents.

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An appendix presents John Z—'s diary in chronological order; this undoubtedly is the most valuable portion of the book. In the body of the work the subject-matter is arranged so as to describe separately "Love at a Distance"; pan-Love towards schoolgirls; friendships with various schoolboys, and John's romance with a girl Elizabeth. This arrangement necessitates considerable redundant reading, hardly compensated for by the author's interpolated critical examination.

The adult, we are told, is specifically incapable of reproducing or re-experiencing the mental state of adolescent friendship-love. Methods of adolescent study based upon retrospection by the psychologist, or examinations of autobiographies, memoirs and works of fiction written by adults, therefore, have little value. A second method of approach, the hypnotic state or the method of free association, gives little support to the author's thesis. It is suggested that the great emphasis laid on the sexual mental experience, which is being made by the representatives of these branches of psychology juxtaposed with their silence as regards the phenomenon of friendship-love, indicates that recollection of this phenomenon does not take place under these conditions.

Direct observation of adolescents is discounted on account of the extreme secrecy they observe in their relationships with grown-ups, especially in regard to these phenomena. The method of choice, therefore, and that adopted here is the investigation of intimate writings spontaneously produced by the adolescent.

The diary form is undoubtedly a valuable means of studying the adolescent. The weakness is that it gives a one-sided view of the facts. John's diary, written during a very narcissistic stage in his development, presents an idealized conception of the self. Anything that does not fit in with this picture is carefully deleted. Anything, for instance, remotely hinting at crude sexuality and in consequence antagonistic to this lofty idealism is referred to as "that"; beyond this the diary tells us nothing.

To form an opinion on adolescent love and sexuality from a study of a diary such as John's is clearly inadequate. It is as if an attempt were made to evaluate post-Elizabethan morality from a reading of one of Fletcher's or Massinger's romantic dramas, in which, by misfortune, the manuscript of the subsidiary plot has been burnt by Mr. Warburton's notorious cook.

In the author's opinion the specific state of adolescent love is one of "great richness and fullness", "an experience of its being something morally positive . . . more than that, something of a sublime nature". There is no conflict between it and the religious moment. Regarding sexuality it is quite otherwise. There are, we are told, four criteria for the sexual mental state, and adolescent love satisfies none of these. First, sexuality is provoked by or directed towards persons of the opposite sex, while "the phenomena of love are experienced by one and the same person, within one and the same time, towards persons of the same and of the opposite sex. (That this cannot legitimately be referred to homosexuality may be argued not only from the fact that the mental state of love is experienced by the same subject towards persons of the opposite sex as well, and that within the same period of time, but also from the fact that the manifestations which we would call sexual are directed—in the same subject—only towards persons of the opposite sex.)" The second criterion is that the fundamental biological end of the sexual mental state is the prolongation of the species, while in the present case love produced a definite repressive effect upon the sexual. As an aside, and in support of this thesis,

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it is argued that during the sexual act the actual state of Love is never being experienced by the mating persons. The third is that there should be a physiological activity of the sexual organs, and there is no evidence in the diary to show the existence of an association between the mental state of love and activity of the sex organs. The last criterion of the sexual is stated to be the mental character of " venereal voluptuousness ", and according to the author's data the mental state of love is absolutely unlike this condition.

Given that the love sentiment is a separate urge arising out of the void during adolescence, it would be helpful to know exactly what, if any, biological purpose it serves. The author of *Friendship-Love in Adolescence* is not particularly convincing in his phenomenological contribution ; perhaps he may be more so in the metaphysical system promised in the near future.

STANLEY M. COLEMAN.

Theory and Practice of Psychiatry. By WILLIAM S. SADLER, M.D. London : Henry Kimpton, 1936. Pp. xxii + 1231. Price 42s.

The amount of energy expended in the production of this massive volume must have been enormous, but it is doubtful if the result justifies the effort. The author has tried to cover a tremendous amount of ground and show the knowledge of some ten or twenty professors. There are a large number of most irritating statements. In Part I, chapter I, we are informed that hormones are " chemical messengers from the ductless glands and other structures, which circulate through the body in the blood stream ". Surely such a statement is much too elementary for a work of such imposing magnitude. We doubt if everyone will agree with the concept of a worship urge on a par with a sex urge and social urge—possibly the psychiatrists of the U.S.S.R. might have some interesting comments to make.

We do not like the use of the expressions "a real hysteric blow-up", a "bit strange ", " getting matters across " to a patient, " McDougallian psychology ".

Does anyone really believe that a sterilization law enforced throughout the United States would result in less than one hundred years in " eliminating a large amount of crime, insanity, feeblemindedness, moronism and abnormal sexuality, as well as many other forms of defectiveness and degeneracy. Thus within a century, our asylums, prisons and state hospitals would be partially emptied . . ." ? And again, "If we should thus conscript our degenerates—sanely classify and properly employ, incarcerate or sterilize them—within a few decades most of our charities, which are dealing largely with problems resulting from feeblemindedness, would go out of business, most of our jails and brothels would be empty . . ." ? The U.S.S.R. we rather think claims to have cured the question of prostitution, but by a less drastic method. We gather that four out of the five " prostitution prophylactoria " which existed in Moscow in 1931 have been closed down, as the problem had largely been dealt with.

The writer gives us a summary of the schools of philosophy on one page. We doubt the value of this in a work of this nature.

There are no doubt many people both in the U.S.A. and this country who go through life enjoying a " good time ", but who would hate to think that they were suffering " from more or less arrested moral development ". Do women really have more pity and sympathy than men, or do they show their sympathy more and men less ?

The writer does not accept the psycho-analytic explanation of religion as a

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regression to infantile attitudes. We must confess to a liking for the subdivision "stupid personalities". The expression "volitional old maid" strikes us as rather original and intriguing. It is also intriguing to read that there are 250,000 of the inhabitants of the United States who are "near" genius or genius—we were beginning to think there was only one! At the same time we do not agree that the possession of an I.Q. of 140 or more entitles one to be called a genius or even a "near" genius. It is a pleasing thought that there are twenty million superior inhabitants of the U.S.A. Does having to be coaxed to play the piano serve as an "earmark of emotional immaturity" in this country?—we hope not for our own sakes! Do the "bully, hayseed, dude, sheik and flapper" *all* have large abdomens and good appetites? And do all efficient men "always have a few minutes' extra time for pleasant social intercourse at breakfast", and do they "stop thinking as soon as they lie down in bed"? Possibly the superior twenty million do. These and many other statements in the book give us very interesting sidelights on life in the United States, the speed at which it moves, and the many maladjustments that must exist and the necessity for dealing with these problems. We would like the author to see life in some of the quieter parts of England. The chapter dealing with personality problems of the pre-school child is very well done, and is followed by other chapters dealing with personality problems which state the problems very well, and give a wide insight into problems which will take generations to deal with satisfactorily.

It is doubtful if any true neurotic suffers from the belief that he or she has committed the unpardonable sin. This is surely over the line into psychosis.

Many of the case-histories have headings that savour too much of the Sunday paper—"A Typical 'Neuro'", "Lifelong Neuroticism", "They Tend to 'Slump'". The psychoses are dismissed in 140 odd pages, which strikes one as a small proportion out of 1200 pages. The author considers that the manic-depressive psychoses are due to endocrine disturbance and that there are two types, one constitutional, the other reactive. He also points out that many "geniuses" are found in the families afflicted with this disorder. The writer tells us that 50–60% of schizophrenics show a family history of mental disorder. We are under the impression that some 66% of normal people show the same if the investigations are carried out thoroughly. Whether the hypoplasia of the cardio-vascular system is due simply to a life of apathy or inactivity is an arguable point. We do not agree with the statement that the most common hallucinations met with in paranoia are auditory. The true paranoiac is not hallucinated at all—a view which the author himself holds on p. 856!

We should have liked to see the treatment of delirium tremens given in more detail. The author mentions Alzheimer's disease, but not Pick's. On the whole the section devoted to the psychoses does not appear at all satisfactory.

There is an extensive section dealing with psychotherapeutics in all its various branches, including "philosophies of life". Medicinal treatment is dismissed in a paltry eighteen lines. The serious student of psychiatry will receive little comfort from the statement—"In case it is deemed necessary to resort to the use of drugs during the course of psychiatric training, aside from the advice already herein offered, the reader is referred to the standard texts on materia medica and therapeutics". We doubt the wisdom of endeavouring to carry out prolonged narcosis or therapeutic hypoglycæmia from "standard texts"!

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There is a very good index, and a comprehensive glossary, but we should hardly have thought it necessary for the twenty million superior adults in the States to be told how to pronounce epilepsy, conflict or idiot!

G. W. T. H. FLEMING.

The Patient and the Weather. By WILLIAM F. PETERSEN. Vol. I. Part II: Autonomic Integration. Michigan: Edwards Bros., 1936. Price $9.00.

This huge volume of 780 pages continues and amplifies the point of view developed in the preceding volumes. The author maintains that his object is to make obvious the close integration between the human organism and the immediate atmospheric environment. He has centred his interest to a considerable degree around the blood-pressure. He begins this volume with an account of constitution, "because so few practitioners of the younger generation have been taught to think of it even in its simplest terms". We doubt if the younger generation ever paid more attention to constitution than they do at present.

The writer promises us that in Volume III he is going to give us the evidence that in the psychoses we deal very probably with (1) a defective substrate, defective in part because of purely genotypic factors, in part probably because of injury early in the segmentation of the embryo and due to meteorological effects on the maternal organism, and (2) with fatigue and injury later in the extra-uterine life, when the defective substrate must meet unduly severe environmental conditions, frequently meteorological. We were afraid this would happen!

The book is full of diagrams and charts showing the enormous amount of work which has been expended.

One wonders whether the pyknic individual during a thunderstorm really suffers from "elements of fright and anxiety due to thunder, lightning, darkness, wind, rain torrents, etc." Surely the human organism was rather better adapted to his environment than this. We doubt whether there is a "well-known association" between the precipitation of acute psychoses and the sudden change in the atmosphere associated with the thunderstorm. When the writer turns his attention to the criminal we find that in the State of Illinois at all events criminals tend to have been conceived in the spring months and fewer in August, which is a distinctly more unstable month.

There is provided a table showing the number of epileptic seizures month by month in a group of 75 patients in the Kankakee State Hospital. The highest number occurred in September and the lowest in June and July. It would be interesting to compare these figures with much larger ones in the British Isles. It is difficult to reconcile the low figure of 6·5 for July with 8·7 for August, and it does suggest that much larger figures properly considered might show that there is little difference between the various months of the year. It is a great pity that there is no index to this book.

G. W. T. H. FLEMING.

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Psychometric Methods. By J. P. GUILFORD. London: The McGraw-Hill Publishing Company, 1936. Pp. xvi + 566. Price 25s.

The author commences: "The primary aim of this volume is to teach the student of psychology how to deal effectively and intelligently with quantitative data." The book is divided into three parts. Part I consists of an introduction to the general theory of errors, including means, standard deviation, the normal distribution, etc., with their applications to psycho-physical methods in general and to various well-known types of experiment in detail. Part II deals with psychological scaling methods and the appropriate calculations. Part III deals with correlation and mental testing; here are explained curve fitting, testing goodness of fit, and simple, multiple, partial and non-linear correlation. The last two chapters deal with the theory and practice of mental testing, and with Spearman's two-factor and Thurstone's vector theories. It concludes with thirteen tables of functions which are commonly required for the work.

The book is closely written and contains a large amount of information. The subjects mentioned above are dealt with in much more detail than the brief list above would suggest. The student who reads and masters the contents should be well abreast of present-day work in psychometrics.

The statistical methods used in the book are deliberately simplified and the student needs little more than school algebra to follow the equations and calculations. In places, however, the simplification appears to have been overdone. Thus, there is no mention of the basic ideas underlying the χ^2 test of goodness of fit. This means that the reader is in great danger of using the χ^2 test in cases where it is totally inapplicable. Simplified statistics always suffer from this danger. We would suggest that the book would be improved by statements, dogmatic if necessary, warning the reader that certain methods are valid only under certain specified conditions.

A second blemish lies in the author's descriptions of tests of significance. He repeatedly describes in full detail the older and rather crude approximations which were once widely used; he then mentions that the accurate method is quite different and proceeds to give details of this. Surely the probable error of a coefficient of correlation should be abandoned for ever now that Fisher's accurate distribution is available.

These are minor blemishes in a book which is comprehensive, detailed and easy to read, and which we can confidently recommend to students of the subject.

W. R. ASHBY.

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Part III.—Epitome of Current Literature.*

1. Anatomy and Physiology.

Left-handedness in the Prehistoric Inhabitants of Alexandria [*Il mancinismo nei preistorici di Castelceriolo (Alessandria)*]. (*Arch. di Antropol. Crim., vol. lvi, p.* 291, *May–June*, 1936.) *Peola. P.*

Prof. Peolo starts by describing two Stone Age implements which could have been used only with the left hand. He refers to prehistoric drawings and statuettes of antiquity, and compared the stages through which children and anthropoid apes pass in the achievement of right-handedness with those depicted in the relics of antiquity. He finds a striking parallel in the following five stages:

(1) Ambidexterity.
(2) Transient period.
(3) Left-handedness.
(4) Transient period.
(5) Predominance of right-handedness.

These stages are intimately concerned with the separation of the prehensile and locomotor functions of the four limbs, and the specialized distribution of these two functions in the upper and lower limbs respectively.

This process of differentiation necessitated the adoption of the upright attitude.

He argues that as man began to fashion tools and weapons, the left hand would be the natural one for defence while the right would be free for more active functions. He chooses the left as the side for defence because of the position of the heart on that side and the ease of access to it in an attack from the left.

H. W. Eddison.

The Thalamus in Relation to the Cerebral Cortex. (*Journ. Nerv. and Ment. Dis., vol. lxxxv, p.* 249, *Mar.*, 1937.) *Walker, A. E.*

The thalamus is divided into three groups of nuclei. The first includes those having entirely subcortical connections, and concerned with either a phylogenetically old system (the nuclei of the mid-line and the nucleus ventralis anterior) or probably intrathalamic associations (the intralaminar nuclei). The second group (the nuclei ventralis lateralis, ventralis posterior, the anterior nuclei and the geniculate bodies) consists of those nuclei receiving fibres from the ascending sensory tracts and projecting to the cerebral cortex. The third group (the nuclei medialis dorsalis, lateralis posterior and pulvinar) receives no fibres from the great ascending pathways, but has numerous connections with the thalamic nuclei of the second group and projects to the association areas (Flechsig) of the cerebral cortex, whereas the fibres of the second group end in the projection centres of the cerebral cortex.

A study of the connections of these cortical areas gives further evidence for the conception that at least a partial synthesis occurs in the nuclei of the third group. The prefrontal area receives fibres from only the adjacent motor areas (4 and 6 of Brodmann), and sends fibres to the latter region and also the parietal cortex.

* A number of abstracts in this section are reproduced from *Chemical Abstracts* by kind permission of Prof. Crane of Ohio University, to whom the Editors wish to express their thanks.

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The fact that the prefrontal cortex has so few connections and yet unquestionably plays a large associative role—as determined by psychological tests, points to its subcortical connections as relaying at least partially synthesized impulses from the thalamus.

The parietal cortex (areas 5 and 7) has a much larger association field—from the prefrontal area, the precentral and post-central convolutions as well as a smaller connection to the periparastriate areas and the temporal cortex. The periparastriate areas likewise have a wide associative field particularly with the striate cortex. It is probable that the thalamic projection to these areas, having such wide cortical connections, must be of a fairly high functional order.

G. W. T. H. FLEMING.

Effect of the Growth Hormone upon the Brain and Brain Weight–body Weight Relations. (*Journ. Comp. Neurol., vol. lxiv, pp.* 469–96, 1936.) *Rubinstein, H. S.*

White rats were injected intraperitoneally with growth hormone for 22 weeks. Control rats were uninoculated or injected with meat extract. The growth hormone produced generalized body growth, but it failed to influence the structural make-up of the central nervous system as judged by studies of the weight, volume, density, water and solid contents of the brain. The growth hormone, therefore, affected the normal brain–body weight relations by decreasing the brain weight–body weight ratio, by lowering the exponent of relation and by leading to a significant deviation from the body–brain growth curves as established by Hatai for the normal. It tended to increase the size of the male pituitary gland but not the female. Extensive bibliography. RACHEL BROWN (Chem. Abstr.)

Metabolism of the Central Nervous System. I : Normal Respiration of the Central Nervous System of the Toad. (*Contrib. Biol. Lab. Sci. Soc. China, vol. xi, pp.* 239–43, 1936.) *Chang, T. H., and Tai, F. I.*

The central nervous system has a high respiration—at least as high as that of the mammalian grey cortex under the same conditions. The respiration parallels alkalinity, shows seasonal variations and is highly affected by oxygen tension.

II : Influence of Electrolytes on its Respiration. (*Ibid., pp.* 243–65.) *Chang, T. H., and Tai, F. I.*

The effects of electrolytes on the respiration of the central nervous system and peripheral nerves are compared. Sodium halides (except NaF, which has depressive effect) all increase its respiration. Lactate, citrate and phosphate cause greater increases. Calcium chloride and KI depress respiration at all concentrations studied. Isotonic KCl gives marked increases which are greater on the brain than on the cord, and, like the normal respiration, it is favoured by alkalinity and seems to require the presence of phosphate. L. T. CHENG (Chem. Abstr.).

Creatine in Brain in the Course of the Ontogenetic Development of Vertebrates. (*Ukrain. Biochem. Journ., vol. ix, pp.* 5–38, 1936.) *Palladin, A. V., and Rashba, H.*

The creatine, water and total-nitrogen contents of the brain during the second half of the embryonic development of rabbits (15th–30th day) are higher than in adult rabbits ; they decrease in later stages. During the first month of post-embryonic development, the creatine and nitrogen contents attain a level characteristic of brains of adult rabbits ; the water content does not attain this level. In embryos of guinea-pigs the same tendency is evident, but not so clearly ; at birth the creatine and total nitrogen contents are the same as in adults ; the water is a little higher. In 3–8-month bovine embryos the contents of creatine, total nitrogen and water are higher in the earlier stages and fall gradually. This process is more evident in the cerebellum ; the water content falls very uniformly.

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Creatine and total nitrogen attain the level characteristic of adult cows at the 6th–7th month of development; at the same time there is yet less dry matter than in the case of adult cows. In different parts of bovine brain there is a difference in the creatine, total nitrogen and water evident long before the beginning of myelinization. The creatine content in the brain of chicken embryos is lower and of total nitrogen and water higher than of adults, and both change at the 18th–19th day, attaining values characteristic of an adult; the water content still remains higher. E. E. STEFANOWSKY (Chem. Abstr.).

Liberation of Acetylcholine-like Substance from the Cut Surface of a Nerve under Physiological Impulses. (*Atti accad. Lincei, vol. xxiii, pp.* 518–21, 1936.) *Bergami, G.*

When the diaphragmatic nerve and vagus nerve of a dog are cut, and the cut end is immersed in Ringer solution for 2 minutes, and then this solution tested on the dorsal muscle of the leech, the muscle contracts. But if the nerve is chilled by holding an ice cube over it for 10–15 minutes and then cut and immersed in Ringer solution, no acetylcholine-like substance is liberated, i.e., no contraction takes place on the leech muscle. A. W. CONTIERI (Chem. Abstr.).

The Physiology and Pharmacology of the Cerebral Vessels. IV: The Action of Adrenaline. (*Arch. Intern. Pharmacodynamie, vol. liv, pp.* 109–28, 1936.) *Bouckaert, J. J., and Jourdan, F.*

Adrenaline causes constriction of the cerebral vessels of the isolated dog brain. In the intact animal rapid injection of adrenaline causes cerebral vasodilatation because of the general rise of blood-pressure. The slow injection of small amounts causes cerebral vasoconstriction.

V: The Action of Carbon Dioxide. (*Ibid., pp.* 155–62.)

By perfusion of the isolated brain carbon dioxide was shown to act directly on the cerebral blood-vessels, causing vasodilatation.

VI: Action of Drugs on the Cerebral Vessels. (*Ibid., pp.* 168–83.)

When an isolated brain preparation was used it was found that posterior pituitary extracts, barium, ephedrine and adrenaline constrict the cerebral vessels. Choline, carbaminoylcholine, amyl nitrite, caffeine benzoate, papaverine, F 883 and F 933 dilate the vessels. The last two substances diminish, and ergotamine abolishes, the vasoconstrictor action of adrenaline.

M. L. C. BERNHEIM (Chem. Abstr.).

The Permeability of the Nerve Centres. I: The Activity and Permeability of the Isolated Cerebro-spinal Axes of Bufo vulgaris. (*Boll. Soc. Ital. Biol. Sper., vol. xi, pp.* 433–4, 1936.) *De Marco, R.*

The amount of potassium eliminated from the cerebro-spinal axes of *Bufo vulgaris* immersed in physiological saline and at complete rest increased markedly as a result of the oxygenation of the nutritive fluid. An increase of potassium elimination took place as a result of electrical stimulation of the preparation; such increase was not influenced, however, by the oxygenation of the Ringer solution. A greater increase in elimination took place through the action of strychnine on the preparation at rest, and greater still on that electrically stimulated.

PETER MASUCCI (Chem. Abstr.).

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2. Psychology and Psychopathology.

A New Series of Perceptual Tests (preliminary communication). (*Brit. Journ. Med. Psych., vol. xvi, p. 97, Nov.*, 1936.) *Penrose, L. S., and Raven, J. C.*

An attempt is made to construct non-verbal tests in accordance with psychological principles. By further adaptations the same tests could be used for blind, deaf or otherwise physically defective subjects. The arrangement of figures in a rectangular pattern indicated to the subject the relationship which has to be discovered.

The structure of the simplest test corresponds to that of verbal problems such as " As black is to white night is to ? ".

The solution of each problem must be unique.

The generalized form of the structure of a test consisting of four elements can be expressed thus :

$$A :: f(A)$$
$$f' :: f'[f(A)].$$

The required answer which is to occupy the bottom right-hand corner can be obtained either by arguing horizontally or vertically. These are three special types of relation possible :

(1) Similarity $f(A) = A$. (2) Opposition where $f(A) =$ not A. (3) Addition where $f(A) = A - X + Z$.

More complicated relations can be expressed by a structure containing nine figures.

In presenting the test to the subject, part of a design is removed and placed among a number of alternative pieces all of the same shape, but bearing either no design or an irrelevant pattern. The designs can be painted in bright colours in order to interest children. The elements in almost every test can be rearranged by permutation. The material therefore has experimental value for studying reliability and the influence of practice, and can be used for purposes of retesting at comparatively short intervals. T. E. BURROWS.

Ego-development and Certain Character Problems. (*Psychoanal. Quart., vol. v, p. 320, July*, 1936.) *Hendrick, I.*

Ego-development is a process which culminates in the capacity of the personality to maintain its existence, and to secure adequate gratification of libidinal and aggressive impulses in a socialized environment. The basic situation of the adult ego is achieved in the pre-œdipal phases of development through identifications which permanently resolve anxiety reactions to primitive hostile impulses. These, in contrast to the identifications by which œdipal and post-œdipal conflicts with the rival are resolved and the super-ego established, are based upon partial, not total, object cathexes.

Each successive group of preœdipal identifications not only provides a solution of the anxiety of the moment, but contributes specific functions to the organization of the developing ego ; each is dynamically related to the type of phantasy and the special organ which dominates during that stage of development.

A failure to resolve an infantile anxiety situation by identification will be represented by a defective ego-function, which necessitates inhibitions or projections that are deleterious to mature object-relationships. These are especially conspicuous in character defects which are recognized clinically as " pregenital ", " narcissistic " or " ego-deficiency " character neuroses.

Repression is a defence against libidinal impulses and aggressions which are highly sexualized ; it is therefore available chiefly to escape anxieties related to passive impulses, to œdipal and post-œdipal activities or their substitutes. Earlier,

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less erotized aggressions, which antidote the splitting of ambivalence between loved and hated objects, provoke retaliation anxiety, and must be defended chiefly by an inhibition, supplemented in some cases by projection or flight from the object into secret though unrepressed play and fantasies.

STANLEY M. COLEMAN.

On Projection. (*Psychoanal. Quart., vol. v, p.* 303, *July*, 1936.) *Feigenbaum, D.*

In the struggle against instinctual drives two groups of defence mechanisms can be distinguished. Projection belongs to the peripheral-active group, in contradistinction to the central-passive one; of this latter group repression, regression, introjection and anxiety are examples.

Projection is the most aggressive mechanism in its group, and the target of this aggression may be either an object-cartharsis (exosomatic projection) or a bodily organ (endosomatic projection). Projection is a preferred method of defence in conditions characterized by a struggle for genitality, such as in convalescence from neurosis, in early phases of psychoses and in delinquency. All forms of projection are reducible to a fundamental defence mechanism appearing in various clinical pictures corresponding to a gamut of specific stages of development, ranging from the normal in dreams, beliefs, superstition, prejudice and creative work, to the pathological in hysteria, hypochondria, phobia, depression and paranoia.

STANLEY M. COLEMAN.

Inhibitions, Symptoms and Anxiety (contd.). (*Psychoanal. Quart., vol. v, p.* 414, *July*, 1936.) *Freud, S.*

Having reduced the development of anxiety to a response to situations of danger, Freud finds that symptoms are created in order to remove or rescue the ego from such situations. If symptom-formation is prevented, then the danger actually makes its appearance, a situation analogous to birth comes about and there are present the first and earliest determinants of anxiety. Symptom-formation has the result of putting an end to this danger situation. It has two aspects: one of them causes in the id that alteration which preserves the ego from danger; the other reveals what it has created in place of the instinctual process thus modified, namely substitute formation. In some cases the defence process is an attempt at flight from the instinctual danger, but in others the ego offers resistance of a far more active kind.

Regarding the situations which occasion anxiety, it is found that every danger situation corresponds to a given stage of development of the psyche to which it appears appropriate. In the adult many of the old causes of anxiety have in reality become inoperative, but only after having first brought neurotic reaction into existence. However, some of these danger situations survive into a later period of life by means of a modification; for example, castration anxiety may persist in the guise of syphilophobia. The neurotic is distinguished from the normal in that his response to these dangers is disproportionately increased, and again because he continues to react with anxiety to situations which should have long ceased to evoke it.

In the final section an attempt is made to find some factor which should make it possible to understand the basis of selection of those individuals who are able to subject the affect of anxiety to normal psychic control or which, on the other hand, determines those who must prove unequal to the task. Freud finds neither Adler's theory of organ inferiority nor Rank's trauma of birth theory an adequate explanation. Here he interpolates a passing regret that it should be so improbable that a physical factor, the isolation of a bacillus or the evolving of some chemical substance, should give a facile solution to this problem. Lastly the psycho-analytic theory of repression, fixation and the repetition-compulsion is reviewed. To this Freud adds three contributory factors which have created the conditions under which the forces of the psyche contend among themselves. Of these the first is a biological one, the protracted helplessness and dependence of the young

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of the human species, bringing into being the first situations of danger, and creating the need to be loved which the human being is destined never to renounce. The second, a phylogenetic factor, is the latency period of sexual development in the human being. The pathogenic importance of this phenomenon accrues from the fact that most of the instinctual demands of infantile sexuality are treated as dangers and guarded against by the ego, so that the sexual impulses of puberty which should be ego-compatible are in danger of succumbing to the attraction exerted by their infantile prototype and of following them into repression. The third, a psychological factor, is to be found in an imperfection of our psychic apparatus which is connected with its differentiation into ego and id. By reason of the dangers which reality offers, the ego is compelled to adopt an attitude of defence towards certain instinctual impulses in the id, and to treat these as dangers. The ego cannot protect itself against internal instinctual dangers as effectively as against a piece of reality which is strange to it. Itself intimately connected with the id, the ego is able to ward off an instinctual danger only by putting restrictions upon its own organization, and by tolerating symptom-formation as a substitute for its crippling of the instinct. If, then, the press of the instinct is renewed, there results for the ego all the difficulties which are recognized as neurotic suffering.

STANLEY M. COLEMAN.

A Case of Agoraphobia. (*Psychoanal. Rev., vol. xxiii, p.* 383, *Oct.*, 1936.) *Greenschpoon, R. R.*

A woman, æt. 42, complained of fear of open places, of being afraid to go out in the street and of gastro-intestinal disturbances. The history showed that towards her dead husband she had conscious feelings of hatred on account of his gambling, alcoholic and promiscuous propensities. Unconsciously she still had strong positive feelings towards him and denied his death. Further, she had very definite feelings of guilt since she held herself in some measure responsible for his suicide. Towards her only daughter conscious maternal affection masked unconscious death wishes on account of the restrictions that the latter put upon her mother's sex life. The phobia was found to have two essential motives. Firstly, to leave home meant for the patient a renewal of the separation from her husband, whose death, for the reasons given above, she denied in her unconscious. Secondly, the phobia was of the nature of a taboo. Sexually abstinent since her husband's death, she had great difficulty in curbing her erotic urges. To go out in the street was unconsciously associated with a prostitution complex. Treatment by Stekel's active analytical technique resulted in removal of the symptoms in six weeks.

STANLEY M. COLEMAN.

Attis: A Study of Castration. (*Psychoanal. Rev., vol. xxiii, p.* 363, *Oct.*, 1936.) *Engle, B. S.*

The Attis theme is traced from its earliest known form, the legends of Pausanius and Arnobius, to its most modern conception in Thomas Mann's novel, *Death in Venice*. In the older versions of the myth Agdistis-Cybele is originally bi-sexual, but the gods perform a surgical operation, cutting off the creature's external male genitals. She is either directly or more remotely the mother of Attis and falls in love with him. The goddess drives Attis mad and in a frenzy he emasculates himself. In remorse for her part in the self-mutilation of Attis she obtains for him a kind of immortality. In later versions and in the Attis cult in Rome the story becomes increasingly symbolical.

The legend supports analytic findings regarding the close association of the castration impulse to the incest motive, identification with the mother and neurotic fear of death. Attis by this act of self-mutilation atones for his incestuous desires, attempts to relive the cycle of his mother, and by the substitution of a part for the whole purchases a form of immortality. STANLEY M. COLEMAN.

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3. Psychiatry.

An Investigation of Deterioration of General Intelligence or "G" in Psychotic Patients. (*Brit. Journ. Med. Psych., vol. xvi, p.* 146, *Nov.* 17, 1936.) *Harbinson, M. R.*

The tests used were the Terman vocabulary test or "V" test which indicates the degree of intelligence before the onset of the illness and the visual-perceptual tests of general ability or "G" tests, which indicate the degree of intelligence at the time of testing. These two series of tests were carried out on some numbers of normal adults. In no case was the "V" score higher than the "G" score. Thirty-six psychotic patients were tested. Of eleven cases of schizophrenia, over 70% showed no deterioration of intelligence, whereas out of 15 cases of melancholia 80% showed deterioration. T. E. BURROWS.

Exogenous Ætiological Factors in Manic-Depressive Psychoses [*Les facteurs étiologiques exogènes dans les psychoses maniaco-mélancoliques*]. (*Ann. Méd. Psych., vol. xv* (*ii*), *p.* 769, *Dec.*, 1936.) *Dickmeiss, P.*

Essentially the manic-depressive psychosis is an endogenous disorder due to definite hereditary congenital anomalies. Nevertheless exogenous factors have a certain importance in the precipitation of this condition. Amongst these provocative factors the writer cites endocrine changes at puberty and particularly at the menopause; psychic trauma; head injuries and cerebral atheroma. A number of illustrative case-histories are included in the text. It is concluded that there is always a pre-existing morbid state, and that one should speak of a reactive depression or mania occurring in persons of a cyclothymic constitution.

Regarding the incidence of manic-depression associated with chronic epidemic encephalitis, the writer only found two cases out of 132 encephalitics examined.

STANLEY M. COLEMAN.

Exogenous Psychoses (*Les psychoses exogènes*). (*Ann. Méd. Psych., vol. xv* (*ii*), *p.* 387, *Oct.*, 1936.) *Rafkin, I. G.*

Having stressed the intimate relationship of exogenous and endogenous precipitating factors and the extreme difficulty in separating the two psychotic groups, two criteria are put forward by means of which the exogenous psychoses may be distinguished.

First a uniformity of reaction evolving in different subjects under the influence of the exogenous reagent and independent of the constitutional type. For example any subject submitted to an exterior action of a certain intensity shows disordered consciousness. The different forms and degrees of this are the most important and characteristic exogenous syndromes. Three types of altered consciousness are described: (i) all states of impoverished thinking and clouding to total loss of consciousness (coma); (ii) delirious states characterized by psychomotor over-activity and the hallucinatory-paranoid syndrome; (iii) the low delirious coma with diminution of consciousness and chaotic psycho-motor agitation. Twilight states without clouding or incoherence are considered to be due to endogenous factors (viz., the epileptic character).

The second criterion is that the reactions and symptom-complexes should only occur under the influence of an exogenous agent and never in an endogenous affection; for example the amnesic syndrome of Korsakov.

Evidence is brought forward to show that in exogenous psychoses schizophrenic symptom-complexes are frequently met without there being necessarily any schizoid anomaly of the personality. On the other hand, the epileptiform syndrome is essentially conditioned by endogenous factors.

Experience with various metallic poisons shows that the exogenous agent may have a specific influence on the endogenous process. Small doses of mercury over a prolonged period are found to activate a schizophrenia in those subjects

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with the constitutional predisposition. On the other hand, chronic lead-poisoning facilitates an epileptic state in those so predisposed. Large doses of the exogenous factor, on the contrary, give rise to reactions and states that are, up to a point, extra-constitutional. STANLEY M. COLEMAN.

The Construction of Depression. (*Int. Journ. of Psycho-Analysis, vol. xvii, p.* 423, *Oct.*, 1936.) *Gerö, G.*

Following a detailed analysis of two cases of depression it is concluded that oral erotism is the favourite fixation-point in the depressive. After the breakdown of infantile genitality the libido regresses to the oral stage under the pressure of the œdipus situation. In these cases oral functions are sexualized, and the sexuality is bound principally to oral conceptions. Oral wishes and feelings of aggression form an upper layer, which must be repressed in order to suppress the whole sadistically tinged sexuality attached to the Œdipus situation. The solution of the oral fixation is attained by making the patient experience the repressed oral impulses, for this experience does not stop at the oral aims, but activates the genital object relation of the Œdipus situation.

Next to the solution of oral fixation the most essential technical question is the making conscious of the aggression. There are with regard to acts of aggression many layers, many interpretations. It is the task of the analysis to make conscious that centre of the feelings of aggression to which the feeling of guilt is most attached. This is always the aggressive impulse originating from the central conflicts of the œdipus-situation.

In the first case studied it was necessary to analyse the obsessional character, the great reserve and rigidity, before the repressed anal-sadistic impulses and especially the oral-sadistic impulses became conscious, which represented here the kernel of œdipal aggression. In the second case the loosening of the harsh super-ego, the disclosing of the sadistic intentions perceptible beneath the manifest masochistic attitude, led at first to diffuse outbreaks of rage, and only after the solution of the oral fixation did the phallic-sadistic impulses appear which had formed the central stratum of the repressed aggression.

STANLEY M. COLEMAN.

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4. Neurology.

Syncope and Convulsions due to a Hyperactive Carotid Sinus Reflex. (*Arch. Int. Med., vol. lviii, p.* 407, *Sept.*, 1936.) *Weiss, S., et al.*

The writers point out that a hyperactive state of the carotid sinus reflex can cause syncope and convulsions resulting from one of the following three mechanisms: (1) cardiac slowing; (2) primary depression of the blood-pressure, and (3) a central reflex to the brain. Attacks can be induced by mechanical stimulation of the carotid sinus. The sinus reflex mechanism is sensitized by digitalis and various morbid states. Both the vagal and depressor type of reaction can be controlled by ephedrine and epinephrine. Atropine abolishes the vagal type but has no effect on the depressor type. The cerebral type is not benefited by these drugs. Surgical denervation of the carotid sinus abolishes spontaneous and induced attacks in suitable cases, but does not influence any of the unrelated accompanying symptoms. Of 10 patients with the cerebral type who were treated by surgical denervation, 8 remained free from attacks. Operation is contra-indicated in patients with severe widespread neurosis associated with a low basal metabolic rate.

It is interesting to note that the response of the carotid sinus in patients suffering from epileptic seizures is normal, and that there is no relation between the behaviour of the carotid sinus reflex and postural hypotension.

G. W. T. H. FLEMING.

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Encephalography with Ethylene. (*Journ. Amer. Med. Assoc., vol. cviii, p.* 461, *Feb.* 6, 1937.) *Newman, H.*

Encephalography with ethylene was carried out in thirty cases, without mortality or serious sequelæ and with radiological results equal to those obtained with air. Although the discomfort of the period of injection is not noticeably lessened, the severity and duration of the post-injection headache is markedly reduced. T. E. BURROWS.

Mechanism of Narcolepsy. (*Journ. Nerv. and Ment. Dis., vol. lxxxv, p.* 424, *April*, 1937.) *Lion, E. G.*

The writer studied the physiology of the neuro-endocrine system of 12 narcoleptics and 12 normals. He found 83% were vagotonic, and the remaining 17% were isoneurotonic. Six narcoleptics showed a vasomotor instability, and 4 had endocrine disturbances. Among the narcoleptics a pyknic body-build was found in 58%. No pure types of sympathicotonic or vagotonic people were found. The writer considers that emotional and other affective disturbances may upset the balance of the autonomic nervous system, producing a more marked vagotonic condition in individuals with a parasympathetic predisposition which precipitates a sleep or cataplectic attack. This would account for the mechanism of narcolepsy on the basis of the pathological physiology of the autonomic nervous system, but the pathways involved await further research into the connections between the hypothalamus and cerebrum. G. W. T. H. FLEMING.

Incidence and Differential Diagnosis of Hypoglycæmic Convulsions. (*Amer. Journ. Med. Sci., vol. cxcii, p.* 600, *Nov.*, 1936.) *Ziskind, E., Hollombe, B. S., and Bolton, R. O.*

The writers draw attention to a hypoglycæmic symptom which calls for special comment, namely that the occurrence of profuse perspiration before an epileptic seizure should strongly arouse suspicion of an underlying hypoglycæmia. Mental confusion for a variable period preceding the convulsion is another symptom suggestive of hypoglycæmia. Variability in the type of attack is in favour of hypoglycæmia.

The relation of the attacks to feeding is very important; the longer the interval since the last meal, the greater the likelihood of the attack being hypoglycæmic. Convulsions occurring only during the early morning or before breakfast should arouse suspicion, especially if the patient has profuse night-sweats or is frequently confused on arising in the morning. Suspicions aroused by the history should be verified by an attempt to produce convulsions through fasting for 36 hours. G. W. T. H. FLEMING.

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5. Pharmacology and Treatment.

Snake Venom (*Moccasin*) *in the Treatment of Epilepsy.* (*Journ. Lab. and Clin. Med., vol. xxii, p.* 572, *Mar.*, 1937.) *Finkelman, I.*

The author treated 8 institutionalized epileptics for 9 weeks with snake-venom. Injections of a 1/3,000 venom solution were given. During the administration of venom the frequency and severity of the seizures were greater than during other periods and the patients were more irritable. There was no correlation between the seizures and the eosinophilic response. G. W. T. H. FLEMING.

The Effect of Intravenous Injections of Sucrose Solution (50%) *on the Cerebro-spinal Fluid Pressure, the Blood-pressure and Clinical Course in Cases of Chronic Hypertension.* (*Amer. Journ. Med. Sci., vol. cxcii, p.* 510, *Oct.*, 1936.) *Murphy, F. D., Herschberg, R. A., and Katz, A. M.*

After the injection of 300–500 c.c. of 50% sucrose solution, there was a prompt and prolonged reduction of spinal fluid pressure followed by a gradual return to

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the basic level in from 8–12 hours. A prompt and profuse diuresis began about 2 hours after the injection and continued for 6 hours. The aggravating symptoms such as headache, vomiting, vertigo, twitching and dizziness were relieved.

G. W. T. H. FLEMING.

Physiologic Effects of Benzedrine and its Relationship to other Drugs Affecting the Autonomic Nervous System. (*Amer. Journ. Med. Sci., vol. cxcii, p.* 560, *Oct.*, 1936.) *Myerson, A., Loman, J., and Dameshek, W.*

The authors carried out investigations on a group of 18 psychotic patients (15 dementia præcox, 2 general paresis and 1 manic-depressive). Benzedrine has a striking and prolonged effect in raising the blood-pressure, together with a less marked effect on the spinal fluid pressure. In some cases the erythrocyte count was raised by 1–3 millions while the leucocyte count was often doubled or even trebled. When used with mecholyl, the blood-pressure lowering effect of the latter drug was at first apparent, followed by an increased blood-pressure when the effects of mecholyl became diminished. When used with atropine the rise in blood-pressure was often extreme.

With amytal, the period of narcosis was shortened and the blood-pressure lowering effect of this drug was diminished. G. W. T. H. FLEMING.

The Central Action of Beta-aminopropylbenzene (*Benzedrine*). (*Journ. Amer. Med. Assoc., vol. cviii, p.* 528, *Feb.* 13, 1937.) *Nattanson, M. H.*

Benzedrine produces in most individuals a definite stimulation of the central nervous system in doses which do not result in a peripheral sympathomimetic action. Euphoria, a feeling of exhilaration, lessening of fatigue, an increased energy and capacity for work and talkativeness follow quite regularly the administration of from 10 to 20 mgrm. Although the drug has its most striking effect in narcolepsy, it acts favourably in states of persistent exhaustion and in individuals who become easily fatigued. The reactions observed indicate that patients who are depressed may be benefited. Studies in normal individuals indicate that benzedrine increases energy and efficiency to a degree that the drug should be of value in the preparation of individuals for activities that require an unusual expenditure of physical and mental energy. T. E. BURROWS.

Physiologic Effects of Acetyl-beta-methylcholine (*Mecholyl*) *and its Relationship to other Drugs Affecting the Autonomic Nervous System.* (*Amer. Journ. Med. Sci., vol. cxciii, p.* 198, *Feb.*, 1937.) *Myerson, A., Loman, J., and Dameshek, W.*

The writers investigated the action of mecholyl in 18 cases of dementia præcox and 2 general paretics, at first alone and subsequently combined with atropine, sodium amytal, benzedrine and adrenaline. 30 mgrm. administered subcutaneously caused flushing of face and chest, perspiration, salivation, rhinorrhœa and lachrymation, a moderate fall in blood-pressure, a rise in pulse-rate and a rise in spinal fluid pressure. All effects except the rise in pulse-rate can be explained on the basis of parasympathetic stimulation. There was no change in the mental reactions or behaviour of the subjects. Atropine quickly overcomes the effect of mecholyl. The combined effect of mecholyl and sodium amytal on the blood-pressure is additive, the resultant fall being very great. Benzedrine, which is a sympathomimetic drug, has its prolonged pressor action temporarily nullified by mecholyl.

G. W. T. H. FLEMING.

The Action of Acetylcholine on the Carotid Sinus. (*Arch. Intern. Pharmacodynamie, vol. liv, pp.* 129–35, 1936.) *Heymans, C., et al.*

Acetylcholine perfused through the carotid sinus of the dog causes a reflex increase in respiration, and a slowing of the heart with a fall in blood-pressure. These effects are not obtained if the nerves from the sinus are cut.

M. L. C. BERNHEIM (Chem. Abstr.).

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Intracarotid Injection of Acetylcholine in Man. (*Rev. Soc. Argent. Biol., vol. xii, pp.* 171–9, 1936.) *Battro, A., and Lanari, A.*

In 17 men 0·02 grm. acetylcholine was injected into a carotid artery. The effects were an immediate violent struggling lasting 30–60 seconds, a hyperpnœa lasting about 30 seconds, a fall in arterial pressure, irregularities in heart-beat for a few minutes, nausea, salivation, homolateral myosis and lachrymation, homolateral sudation and a homolateral vasodilation lasting 10–15 minutes. The last-named effect showed a sharp dividing line down the exact centre of the face.

L. E. GILSON (Chem. Abstr.).

The Comprehensive Management of Delirium Tremens. (*Journ. Amer. Med. Assoc., vol. cviii, p.* 345, *Jan.* 30, 1937.) *Piper, P., and Cohn, J. V.*

Three hundred consecutive cases of delirium tremens were treated by this method, with a resultant crude mortality of 5·3% and an average stay in hospital of 4·8 days.

(1) No alcohol is given the patient from the time of entering the hospital.

(2) Absolute rest in bed under close observation.

(3) Extract of cascara 10 gr. is given on admission, followed by 1 oz. of magnesium sulphate 2 hours later.

(4) Magnesium sulphate 1 oz. is given every morning for 3 days unless there have been more than 4 bowel movements on the previous day.

(5) Alkalis in the form of imperial drink are given 3 times daily.

(6) Spinal fluid drainage is done as soon after admission as possible and may be repeated as often as indicated.

(7) 50 c.c. of 50% dextrose is given intravenously from 2 to 4 times a day.

(8) 10 c.c. of 50% magnesium sulphate is given intramuscularly once to twice a day for 2 days.

(9) Caffeine sodium benzoate 7½ gr. is given every 4 hours for 6 doses.

(10) The patient is digitalized in from 36 to 48 hours and is then placed on a maintenance dose.

(11) Paraldehyde 3 or 4 drm. is given from 1 to 3 times a day for sedation. Intravenous sodium amytal may be used, but not more than 15 gr. should be given to any one patient.

(12) Hydrotherapy is indicated where possible.

(13) A high caloric soft or liquid diet, supplemented by vitamin-containing substances, especially of the B group, is ordered.

Gastric lavage with sodium bicarbonate is indicated in cases of vomiting.

(14) Fluids are given according to the patient's desire.

(15) Should the patient be asleep he is not awakened for any reason, medicinal or otherwise.

T. E. BURROWS.

Treatment of Menstrual Migraine. (*Journ. Amer. Med. Assoc., vol. cviii, p.* 612, *Feb.* 20, 1937.) *Moffat, W. M.*

The result of treatment with gonadotropic factor (follutein) extracted from the urine of pregnancy in 17 cases of menstrual migraine followed over a period of 3 years was relief in all. In only 4 of the 11 cases studied rœntgenologically was a normal sella turcica found. These 17 women were all menstruating more or less regularly. The results tend to confirm the hypothesis that a leading factor in the production of menstrual migraine is an ovarian hypofunction, perhaps associated with an anterior pituitary hyperfunction.

T. E. BURROWS.

Nitrogen-alkyl Barbituric Acid Derivatives. (*Journ. Amer. Pharm. Assoc., vol. xxv, pp.* 858–9, 1936.) *Swanson, E. E.*

Fourteen new nitrogen-alkyl substituted barbituric acid derivatives with the general formula

$$CO.NH.CO.NR.CO.CR'R''$$

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(in which R = Me or Et, R′ = Am, l-methylbutyl, iso-Am, iso-Bu, *sec*-Bu or l-methylpentyl, and R″ = Me, Et or allyl) were studied by injecting solutions of the sodium salt of the compounds intraperitoneally into albino rats weighing 75–125 grm. (av. 97 grm.). Substitution of an Et or Me group in place of the H on the N distinctly shortens the duration of action. With an Et in place of Me on the N, the anæsthetic and the lethal dose in mgrm. per kgrm. was more than twice those of the Me group on the N; but no change in the duration of action was observed. Duration of action is therefore not dependent on the quantity of drug administered. A. PAPINEAU-COUTURE (Chem. Abstr.).

Synergism and Antagonism of Drugs. II. The Action of Physostigmine on Autonomic Ganglia. (*Journ. Pharmacol., vol. lviii, pp.* 105–10, 1936.) *Koppanyi, T., Dille, J. M., and Linegar, C. R.*

Physostigmine abolishes the vagal (parasympathetic) paralysis of nicotine and curare and the sympathetic paralysis of nicotine. The ganglionic paralytic effect of nicotine and curare in the autonomic system is opposed by physostigmine but not by pilocarpine. Atropine prevents the ganglionic effect of physostigmine. These actions of physostigmine are interpreted as being due to a stimulation of the release of acetylcholine, previously interfered with by nicotine or curare.
T. H. RIDER (Chem. Abstr.).

Atropine in the Cure of Postencephalitic Parkinsonism. (*Argomenti Farmacoterap., vol. iv, pp.* 11–14, 1936.) *Budinis, I.*

A report of the administration of gradually rising doses of atropine sulphate in 50% solution according to the method of Römer in 9 cases of Parkinsonism with favourable results, which are discussed. C. R. ADDINALL (Chem. Abstr.).

The Respiratory Centre and Narcosis. (*Arch. Intern. Pharmacodynamie, vol. liv, pp.* 219–46, 1936.) *Mansfeld, G., and Tyukody, Fr. v.*

Four active respiratory centres and one inhibitory centre are concerned in respiration. The cerebellar dog breathes normally. Removal of the cerebellum causes cessation of respiration. If the upper region of the medulla is then removed respiration begins again. This region thus contains an inhibitory centre, and after its removal the respiration is no longer affected by oxygen or carbon dioxide. The pons also contains a respiratory centre whose function is similar to that in the cerebellum, but reflexes from the carotid sinus act only on the cerebellar centre. The lower region of the medulla contains two respiratory centres. Phenobarbital has a different affinity for the different centres. If only one centre is regulating respiration the all-or-none law holds for the action of this drug.
M. L. C. BERNHEIM (Chem. Abstr.).

The Brain Content of Anæsthetics in Experimental States of Hypo- and Hypersensibility. (*Anesthésie et Analgésie, vol. i, pp.* 229–42, 1935.) *Tiffeneau, M.*

Acidosis favours hypnosis. In fish, the rapidity of narcosis is greater in an acid medium. In rabbits chloroform is more rapidly absorbed into blood and brain by animals in a state of acidosis. In dogs and rats the brain content of alcohol is greater in normal animals than in those which have acidosis. In mice the relative amounts of anæsthetic C_2H_5Br fixed by the blood and brain vary with the rate of production of anæsthesia. The right hemisphere apparently contains more C_2H_5Br than the left, and the posterior portion of the brain contains more than the anterior. T. H. RIDER (Chem. Abstr.).

Barbiturates. XVII. The Effect of Prolonged Chloroform Anæsthesia on the Duration of Action of Barbiturates. (*Journ. Pharmacol., vol. lviii, pp.* 119–27, 1936.) *Koppanyi, T., Dille, J. M., and Linegar, C. R.*

Chloroform anæsthesia (2 hours) of cats and rabbits prolongs and deepens the anæsthesia from pentobarbital and barbital 24 hours later. Conclusion: The

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chloroform, while completely eliminated, has injured the cells of the central nervous system, making them more susceptible to barbiturates.

XVIII: A Peripheral Action of Barbiturates. (*Ibid., pp.* 128–34.) *Linegar, C. R., Dille, J. M., and Koppanyi, T.*

Amytal, pernocton, moderate doses of pentobarbital and large doses of barbital may paralyse the peripheral vagus but not the central vagus, whereas phenobarbital has no paralytic effect. Pilocarpine and acetylcholine still slow the heart. Vagus excitability is restored by physostigmine. Conclusion: The barbiturate action is in the peripheral ganglionic cells of the heart.

XIX: The Barbiturate-picrotoxin Antagonism. (*Ibid., pp.* 199–228.) *Koppanyi, T., Linegar, C. R., and Dille, J. M.*

A review and extension of data. Metrazole is of value in mild cases of barbiturate poisoning, picrotoxin also being required in the treatment of more severe poisoning. T. H. RIDER (Chem. Abstr.).

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6. Pathology and Biochemistry.

Effect of Encephalography on Blood-sugar Level of Children. (*Amer. Journ. Med. Sci., vol. cxciii, p.* 259, *Feb.*, 1937.) *Bradley, C.*

The writer found that encephalography as usually carried out in children consistently resulted in hyperglycæmia with blood-sugar levels often in the vicinity of 200 mgrm.%. The blood sugar rises rapidly during spinal fluid replacement, reaches its peak within an hour and falls over a period of several hours. This hyperglycæmia bears no evident relationship to the anæsthetic used, the clinical condition or age of the child studied, the amount of fluid withdrawn or air injected.
G. W. T. H. FLEMING.

Vital Staining of the Central Nervous System (*Sulla colerazione vitale del sistema nervoso*). (*Riv. di Neur., vol. ix, p.* 253, *Oct.*, 1936.) *Giordano, F.*

After injecting during life mercuric chloride and cantharidin, the author studied their effects upon the choroid plexus in rabbits. He found, as others before him, that cantharidin selects the connective tissue of the villi, while corrosive sublimate attacks the epithelium. He describes and discusses the lesions found.
H. W. EDDISON.

Creatinuria in Post-encephalitic Parkinsonism [*Sul ricambio dei corpi creatinici nel parkinsonismo post-encefalitico*]. (*Riv. Sper. di Freniat., vol. lx, p.* 388, *Sept.*, 1936.) *Porta, V., and Pelliecioli, V. G.*

The authors state that they have frequently found creatinuria in post-encephalitic cases. They consider that treatment with atropine reduces the amount of both creatine and creatinine in proportion to the degree of improvement obtained.
H. W. EDDISON.

The Iso-electric Point of Protein in the Cerebro-spinal Fluid [*Il punto isoelettrico della proteina del liquido cefalo-rachidiano*]. (*Riv. di Neur., vol. ix, p.* 368, *Oct.*, 1936.) *Tornu, A.*

The iso-electric point of the total protein offers difficulties owing to the different value of each individual protein and variations in their proportion.

The highest degrees of precipitation are seen in general paralysis, acute encephalitis and tubercular meningitis. In G.P.I. the zone of precipitation reaches its maximum at pH 5·1–5·4. In meningitis it tends towards the acid side with maximum precipitation round about pH 4 7. In normal and in non-inflammatory cases the zone of precipitation is narrowly limited to about 4·7. The readings are not constant in tabes. H. W. EDDISON.

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Diabetes Insipidus (*Il diabete insipido*). (*Riv. di Neur., vol. ix, p.* 263, *Aug.*, 1936.) *Salmon, A.*

The author discusses the three fundamental factors in the ætiology of the disease:

(1) The polyuric centre in the diencephalon.

(2) The pituitary factor in the posterior lobe. He refers to the frequency of polyuria in acromegaly, the adiposogenital dystrophy, etc.

(3) The suprarenal factor, influencing the saline content of the urine, the arterial tension and other functions. H. W. EDDISON.

Magnesium Salts in Treatment [*Sali di magnesio in terapia psichiartica*]. (*Rass. di Studi Psichiat., vol. xxv, p.* 788, *Sept.–Oct.*, 1936.) *Mossa, G.*

Magnesium sulphate in doses of 10 c.c. of a 10% or 15% solution is injected intravenously. It is said to be particularly useful in the excitement of manic-depressive cases and epileptics. Anæsthesia for pain and temperature, muscular relaxation and abolition of superficial reflexes are transient results of the injections. The systolic blood-pressure falls slightly, especially in cases with hypertension, but the diastolic pressure is hardly affected. These signs disappear in about an hour, but the patient remains calm and quiet, though fully conscious all the time until normal sleep follows. No ill-effect has been noted.

H. W. EDDISON.

The Orthophosphoric Acid Reaction in the Cerebro-spinal Fluid [*La reazione all' acido ortofosforico sul liquido encefalo-rachidiano*]. (*Rass. di Studi Psichiat., vol. xxv, p.* 714, *Sept.–Oct.*, 1936.) *Galli, G. M.*

The reaction in the cerebro-spinal fluid is found to be positive in 90% of syphilitic cases, but it is by no means specific. It may be positive in disseminated sclerosis, meningitis, peripheral neuritis and other conditions.

The material investigated consisted of 206 fluids from various neurological and psychiatric cases. H. W. EDDISON.

The Connective Tissue Reaction in Multiple and Diffuse Sclerosis. (*Arch. of Path., vol. xxiii, p.* 338, *Mar.*, 1937.) *King, L. S.*

The writer found in every one of 13 cases of multiple sclerosis networks of argyrophil connective-tissue fibres growing diffusely into the parenchyma. The extent of this growth varied from case to case and even from plaque to plaque. In part the reticulin nets grew from blood-vessels of small calibre, predominantly capillaries and precapillaries, in part they appeared to grow independently of pre-existing reticulin. Diffuse reticulin invasion may be one of the early pathological reactions in multiple sclerosis, and is found not only in the white matter but in the cerebral cortex and other grey masses. The growths are related to the disintegration of myelin, with the intensity of the process playing some role. Such reticulin nets bear no correlation with the degree of gliosis, of axis-cylinder destruction or of perivascular infiltration.

Eleven cases of diffuse sclerosis were divided into three groups, of which the first showed no reticulin nets, the second a slight or moderate growth similar to that in multiple sclerosis, and the third a profound growth, showing qualitative as well as quantitative differences from that in multiple sclerosis.

G. W. T. H. FLEMING.

Lesions of the Nervous System of the Rat in Vitamin B Deficiency. (*Arch. of Path., vol. xxiii, p.* 207, *Feb.*, 1937.) *Davison, C., and Stone, L.*

Diets deficient in vitamin B_1 or in vitamins B_1 and B_2 (totally and subtotally deficient) result in the rat in neurological signs, such as dragging and paralysis of the extremities, equilibratory disturbances, priapism, convulsions and tonic retractions of the head. Pathologically the outstanding picture is that of disintegration of

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the myelin sheaths of the peripheral nerves, and vacuolation and liquefaction necrosis of the ganglion cells of the mesencephalon, metencephalon and anterior horns of the spinal cord. Hæmorrhages with changes in the ganglion and glia-cells of the mesencephalon are also met with in the animals with convulsions.

The pathological changes in the nervous system in animals suffering from inanition are essentially the same as those in animals deprived of vitamin B_1 or of both vitamins B_1 and B_2. If anything, the changes in the peripheral nerves in animals subjected to inanition are more marked. In such animals the pathological changes are essentially the same whether the animals are totally starved or are given abundant vitamin while starving. The clinical manifestations in animals subjected to inanition, however, are not identical with those in animals suffering from vitamin B deficiency, possibly because of the rapid death of the starved animals. G. W. T. H. FLEMING.

Blood Cholesterol and the Manic-Depressive Psychosis. (*Journ. Lab. and Clin. Med., vol. xxii, p.* 240, *Dec.*, 1936.) *Schube, P. G.*

The author investigated 71 cases of manic-depressive psychosis and 53 normals. He took the normal range to be between 110–195 mgrm., with a mean value of 148·1·7 and a median value of 150 mgrm. The range among the manic-depressives was from 50–428 mgrm. with a median value of 104·6. Only 23·8% had a blood-cholesterol value between normal levels.

In the separate groups, 84·8% of the 33 manic cases showed a cholesterol value below normal limits; the remaining 15·2% were within normal limits. In the depressive group; practically one-third were below normal, one-third normal and one-third above normal. The author thinks it is possible that some disturbance of the balance between the endocrine glands, the autonomic nervous system and the emotions is the factor at work. G. W. T. H. FLEMING.

Cerebral Lesions in Hypoglycæmia. (*Arch. of Path., vol. xxiii, p.* 190, *Feb.*, 1937.) *Baker, A. B., and Lufkin, N. H.*

The writers studied the brains of three patients who died in a hypoglycæmic state. They found numerous new and old hæmorrhages scattered irregularly, which were most numerous in the patients who had had the most severe convulsions. The ganglion cell changes found were due entirely to post-mortem changes. They carried out experiments on six rabbits, but could find no evidence of cell alterations of pathological significance. G. W. T. H. FLEMING.

Evidence of Selection in the Building up of Brain Lecithins and Cephalins. (*Journ. Biol. Chem., vol. cxviii, p.* 131, *Mar.*, 1937.) *McConnell, K. P., and Sinclair, R. G.*

By feeding young rats with elaidic acid both before and after birth, the elaidic acid content of the fatty acids in the lecithins and cephalins of the brain was found to be only about one-fourth of that of the liver and muscles. Thus there would appear to be a greater degree of selection in the building up of brain phospholipids than those of liver and muscle. G. W. T. H. FLEMING.

The Carbohydrate Metabolism of Brain. I: The Determination of Glycogen in Nerve Tissue. (*Journ. Biol. Chem., vol. cxvi, p.* 1, *Nov.*, 1936.) *Kerr, S. E.*

The writer describes his modification of the Pflüger procedure for the estimation of glycogen. The method depends on (*a*) avoidance of post-mortem change during preparation, (*b*) rapid solution of the tissue by digestion with hot alcoholic potassium hydroxide, (*c*) separation of cerebrosides by means of hot methyl alcohol—chloroform mixture, and (*d*) correction for non-fermentable reducing substances liberated during acid hydrolysis. By this method the recovery of glycogen added to brain averaged 95·6%. Values of from 70–130 mgrm. per 100 grm. were found in mammalian brains frozen in situ with liquid air.

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II: The Effect of Varying the Carbohydrate and Insulin Supply on the Glycogen, Free Sugar and Lactic Acid in Mammalian Brain. (*Ibid., p.* 9.) *Kerr, S. E., and Ghantus, M.*

The glycogen content of normal well-fed and fasting dogs lies between 77–150 mgrm. per 100 grm., and between 70–99 mgrm. in rabbits. Fasting, over-feeding, glucose infusion with or without insulin, phloridzin poisoning followed by adrenalin and pancreatectomy all failed to cause significant changes in the brain glycogen. Overdosage with insulin caused a marked decrease in the brain glycogen of dogs and rabbits. The free sugar of brain in the control animals varied from 35–75 mgrm. per 100 grm. in rabbits and from 45–86 mgrm. in dogs. Lowering of the blood sugar by phloridzin poisoning or by insulin caused a corresponding decrease in brain sugar. Hyperglycæmia caused by pancreatectomy or by administering glucose caused a rise of sugar in the brain. The free sugar of the brain was constantly lower than that of blood, except in extreme insulin hypoglycæmia. Neither lactic acid nor phosphocreatine of brain was affected by any of the experimental conditions. G. W. T. H. FLEMING.

The Effect of Acetylcholine and other Constituents of the Adrenal Gland upon Blood Sugar and Amino-acids. (*Amer. Journ. Physiol., vol. cxvii, pp.* 542–52, 1936.) *Davis, B. L., jun., and Luck, J. M.*

Acetylcholine induces hyperglycæmia in rabbits if convulsions intervene. In the absence of convulsions it causes a moderate degree of hypoglycæmia. Acetylcholine hyperglycæmia is obtained after destruction of the adrenal medulla. It is, therefore, not due to a discharge of adrenaline. Acetylcholine causes hyper-amino-acidæmia both in normal rabbits and after destruction of the adrenal medulla. Cortin and ascorbic acid are without effect on either blood sugar or amino-acid nitrogen. The minimum hyperglycæmic dose of adrenaline is slightly less than the minimum dose required for the production of hypo-amino-acidæmia.
E. D. WALTER (Chem. Abstr.).

The Effect of Emotion on the Blood Cholesterol. (*Z. Ges. Exptl. Med., vol. xcviii, pp.* 428–31, 1936.) *Dobreff, M., Peneff, L., and Wittkower, E.*

Anxiety increases the cholesterol contents of serum. Samples taken during hypnosis are usually lower and those just before operation higher than controls.
MILTON LEVY (Chem. Abstr.).

Fat Metabolism in the Nerve Tissues of Pigeons Affected by B Avitaminosis. (*Journ. Biochem.* [*Japan*], *vol. xxiv, pp.* 153–205, 1936.) *Takato, Y.*

Polished rice contains 2·4 grm. total lipide and 0·03 grm. total cholesterol per kgrm. No lecithin or cephalin could be demonstrated. The analytical methods employed could determine gravimetrically 9 mgrm. total fatty acid in 0·2 grm. brain-tissue and 1–2 mgrm. total cholesterol in 0·1 grm. tissue; furthermore, 0·05 mgrm. phosphatide phosphorus could be determined in 0·2 grm. and 0·04–0·08 mgrm. nitrogen in 0·5 grm. lipide material. In fasting pigeons there was a decrease in total fatty acid of the brain as compared with that of normally nourished birds. In pigeons on diets free from vitamin B_1 and B_2 there was also a decrease in total fatty acid content which was due to the fasting, but in the group lacking B_1 the decrease was greater than in ordinary inanition. No differences were observed in the different groups of pigeons so far as total cholesterol, phosphatide phosphorus or amino-nitrogen of the lipide material are concerned. The lipide of the spinal cord was not materially affected by fasting, but a decrease was found in the pigeons lacking $B_1 + B_2$ or only B_1 in their diets. In the peripheral nerves no marked difference was noted between the starved and normally fed groups of pigeons, but a decrease in total fatty acids was found in the pigeons on a diet lacking B_1 and B_2, and especially on the B_1-free diet. Vitamin B_2 stimulates the oxidation of fats and B_2 inhibits it in the nerve tissues. S. MORGULIS (Chem. Abstr.).

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The Acetylcholine Metabolism of a Sympathetic Ganglion. (*Journ. Physiol., vol. lxxxviii, pp.* 265–83, 1936.) *Brown, G. L., and Feldberg, W.*

Acetylcholine liberated in a ganglion by preganglionic stimulation can, if protected by eserine, reach such a concentration that it produces a partial paralysis of the ganglion-cells. Synthesis of acetylcholine appears to occur during a stimulation of the preganglionic fibres. This synthesis is not affected by eserine. Prolonged stimulation does not significantly alter the amount either of acetylcholine or of choline extractable from a ganglion. E. D. WALTER (Chem. Abstr.).

A Contribution to the Study of Polypetidæmia (*Contribution à l'etude de la polypeptidémie en clinique neuro-psychiatrique*). (*Ann. Méd. Psych., vol. xv* (*ii*), *p.* 572, *Nov.*, 1936.) *Artur, Boucher et Coulonjou.*

In 14 out of 27 investigations of the polypeptide level in the blood, there was a hyperpolypeptidæmia between 0·043 and 0·090 grm. Of these 14 cases 11 could be explained by an associated peptic insufficiency. In 2 cases mental improvement was paralleled by a lowering of the polypeptide level. No direct association was found between hyperpolypeptidæmia and any specific neurosis or mental disorder. The majority of the cases studied had alcoholic psychoses.

Sixteen investigations of the polypeptides in the cerebro-spinal fluid showed no abnormally high level; this group included a number of cases in the acute stages of delirium tremens. STANLEY M. COLEMAN.

Contribution on the Origin of Increased Protein in the Cerebro-spinal Fluid of Tumours of the Central Nervous System. (*Journ. Nerv. and Ment. Dis., vol. lxxxv, p.* 373, *April*, 1937.) *Deane, J. S.*

The writer investigated the data provided by 81 consecutive cases of tumour within the intracranial or intraspinal cavities. He found that the presence of spontaneous hæmorrhage or necrotic products or both had little if any relation with an increased cerebro-spinal fluid protein. Generalized increased intracranial pressure *per se* appears to have no relation with increased protein, neither does the histological type of the tumour.

Three types of internal hydrocephalus are distinguished according to their modes of origin and the amount of cerebro-spinal fluid protein in each type:

(*a*) Internal hydrocephalus due to partial or complete obstruction of the venous effluence of the choroid plexus, in which the rate of fluid formation exceeds the rate of absorption. In this type the fluid protein is usually increased.

(*b*) Internal hydrocephalus due to interference with the absorption of fluid, in which its formation continues although the rate is decreased. This type is usually associated with a normal or often decreased fluid protein.

(*c*) In this type a combination of both *a* and *b* exists. The amount of fluid protein may be variable, but is usually increased.

The increase in fluid protein appears to be due almost exclusively to increased transudation of blood-plasma proteins into the fluid, which occurs because of a partial or complete obstruction to venous return flow.

Increased fluid protein in intracranial tumours is apparently caused by local compression of relatively non-anastomotic venous channels draining the choroid plexuses, especially those of the lateral and third ventricles.

G. W. T. H. FLEMING.

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CONTENTS FOR MARCH, 1937

PAGE

Original Articles—

The Electro-Encephalogram in Epilepsy; by *F. Golla F.R.C.P., S. Graham, M.B., B.S., D.P.M., and W. Grey Walter, M.A.Camb.* 137

The Differentiation of Neuroses and Manic-Depressive Psychoses; by *Desmond Curran, M.B.Camb., M.R.C.P.Lond., D.P.M.* 156

The Enumeration of the Blood-Platelets in Mental Disorders; by *D. K. Bruce, M.B., Ch.B., D.P.M.* 175

A Case of Hysteria showing Spontaneous Hyperventilation Tetany; by *Russell Fraser, M.B., N.Z., M.R.C.P.Lond.* 190

The Effect of Altering the Conditions of the Autonomic Nervous System on the Choline Esterase Level in Human Blood Serum; by *Maxwell Shaw Jones, M.B., M.R.C.P.Edin., D.P.M., and Henry Tod, B.Sc., Ph.D.* . 202

The Urea Clearance Test in Psychotics; by *Andrew M. Wyllie, M.B., Ch.B., B.Sc.Glas., Dipl. Psych. Edin.* 208

A Note on the Use of Diathermy in Idiopathic Epilepsy; by *J. L. Clegg, M.D., D.P.H., D.P.M.* 216

Reviews 218

A Study of Hypoglycæmic Shock Treatment in Schizophrenia; by *Isabel G. H. Wilson.*—An Enquiry into Prognosis in the Neurosis; by *T. A. Ross, M.D., F.R.C.P.*—Inhibitions, Symptoms and Anxiety; by *Sigmund Freud.*—Friendship-Love in Adolescence; by *N. M. Jovetz-Tereshchenko.*—Theory and Practice of Psychiatry; by *William S. Sadler, M.D.*—The Patient and the Weather; by *William F. Petersen.*—Psychometric Methods; by *J. P. Guilford.*

Epitome of Current Literature 229

1. Anatomy and Physiology.—2. Psychology and Psychopathology.—3. Psychiatry.—4. Neurology.—5. Pharmacology and Treatment.—6. Pathology and Biochemistry.

CONTENTS OF SUPPLEMENT

The Royal Medico-Psychological Association: Quarterly Meetings.—South-Eastern Division. — South-Western Division. — Northern and Midland Division.—Scottish Division.—Irish Division.—Midland Mental Pathological Society.—Research and Clinical Committee.—Obituary: D. M. Cassidy.——Board of Control.—Bureau of Human Heredity.—Notices by the Honorary Librarian.—Notices by the Registrar.—Notices by the General Secretary.—Notices of Meetings.—Notice by the Secretary, Research and Clinical Committee.

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THE
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Part I.—Original Articles.

AN INQUIRY INTO THE INCIDENCE OF "NEUROPATHIC" CONDITIONS IN THE RELATIVES OF NORMAL PERSONS.*

REPORT BY THE MENTAL DEFICIENCY COMMITTEE OF THE ROYAL MEDICO-PSYCHOLOGICAL ASSOCIATION.

THERE have been several inquiries into the incidence of various "neuropathic" conditions among the relatives of the mental defective, and it has been found that mental defectives often have relatives who have suffered from such conditions as cerebral hæmorrhage, epilepsy, alcoholism, etc. Thus it has been stated that 80% of mental defectives have a "positive" family history, and it is suggested that this figure (80%) supports the theory that most mental deficiency is due to hereditary factors. Before the significance of such a figure can be discussed we must first know the corresponding figure for the normal population.

In order to attempt to ascertain the frequency of such abnormalities among the relatives of the normal person, the Mental Deficiency Committee devised a questionnaire which was circulated among the mental hospitals, and it was requested that it should be filled in by as many of the staff as possible.

The questionnaire asked for information about the following relatives: siblings, parents, uncles and aunts, nephews and nieces, children, grandparents.

The numbers of such relatives were recorded and columns were provided for marking the presence of the following abnormalities:

(A) Fits.
(B) Paralysed
(C) Deaf and dumb } from childhood.
(D) Blind
(E) Insane.

* The inquiry was entrusted to a sub-committee consisting of the following members: Drs. W. R. Ashby, C. J. C. Earl, E. O. Lewis, L. S. Penrose and R. M. Stewart. The sub-committee wishes to express its appreciation of Dr. Ashby's services in tabulating the data and preparing the Report.

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(F) Weak-minded.
(G) Rather peculiar.
(H) Alcoholic.
(J) Tubercular.
(K) Mental condition normal.
(L) ,, ,, not known.

It was appreciated that informants might hesitate to fill in a full account of the abnormalities in their family. The form was therefore arranged so that only figures or crosses need be put in the form, and every care was taken to ensure anonymity. Each form was accompanied by a stamped addressed envelope.

Fifteen hundred forms were distributed. Of these, 415 were returned completed. These 415 forms were next gone through in detail and a few minor corrections were made.

Each person was allowed to come into only one disease class. For instance, one form showed a grandfather as being "insane" and also "rather peculiar". In such cases the less severe condition was deleted. It appeared necessary to restrict each person to only one class, as otherwise we should merely be collecting a list of his symptoms. This occurred in only a few cases, and could not affect the results appreciably.

The first point for examination is to ascertain the number of normal people (informants) who have a "positive" family history. Of the 415 informants, 235 were marked as having one or more relatives with the abnormalities listed at the beginning. This number corresponds to 57% (Table I). (The tables have been collected together in Appendix I.) This is the figure which the questionnaire was primarily designed to ascertain. The practical sampling limits for this figure are 52 to 61%.

That the result should come out as high as 57% is clearly interesting. The committee are unanimously agreed that this may be accepted as a *lower limit* to the true figure. That is to say, they are satisfied that the figure which would be obtained by personal investigation with reliable informants, and covering the family history more exhaustively, would certainly be higher than this. There were several factors in the investigation which would tend to depress the figure, while there appeared to be no appreciable factor which would make it incorrectly high. The main factors tending to give a falsely low figure are:

(1) The tendency of nurses with a "bad family history" to refuse to fill in the forms. This is undoubtedly an important point. Several cases where this had happened were known personally to members of the committee.

(2) The fact that information was not asked about all possible relatives. Were the inquiry to be extended to further relatives, this could only result in a raising of the figure.

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(3) The fact that the inquiry did not include all conditions which might be considered to be of " neuropathic " importance.

After taking these facts into consideration there can be little doubt that the incidence of a " positive " family history in the normal population must be at least 57%.

A fact which supports this conclusion is that one member of the committee was able to obtain twenty forms which he knew to be reliable. The incidence of abnormalities in this series was slightly but significantly higher than in the rest of the series.

A further point of interest is that no less than 23% of the informants had a relative who suffered from a disorder marked A–F in the list above. These disorders (" fits ", " insane ", etc.) were of a type not likely to be filled in incorrectly, especially as most of the informants were on the staff of mental hospitals. The practical sampling limits of this figure are 19% to 27%. It is interesting to find that at least a fifth of the normal population give a family history with a major neurological or psychiatric abnormality. The committee is satisfied that this figure is likely to be in error only by being too low.

Since the report was completed, we have discovered a reference to some similar work by Berlit. He also examined the family histories of the staff of a large mental hospital in Saxony.

He collected details about 362 members of the staff, with their sibs and parents, the total being 2,893 people.

His figures are not exactly comparable to ours, since he classified the results somewhat differently, but by dissecting out some of his figures it is possible to obtain a reasonably good comparison involving the same types of abnormalities.

Among the sibs of the informants there were 13% of people who had abnormalities corresponding to our list. In our series the figure was 6%. In the parents he found 15%. We found 6%. In each case his figure is significantly larger than ours.

Details for other relatives were not given in his paper.

His method of collecting the facts should be mentioned. He was in general practice, but attended the whole staff medically. His data were obtained by personal interview, and were therefore likely to be more reliable than ours.

An exact comparison is impossible, but his results support our contention that our figures are likely to be in error only by being too low.

Family Size and the Presence of Abnormalities.

A second question on which the forms will give some information is in regard to the general question of whether the so-called " degenerate stocks " are breeding faster than the more normal. There is no accepted definition either of " degenerate stocks " or of " rapidity of breeding ", and consequently the question may be approached statistically from a number of points of view.

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The general method will usually be to compare family size in those families with abnormalities with family size in the more normal families.

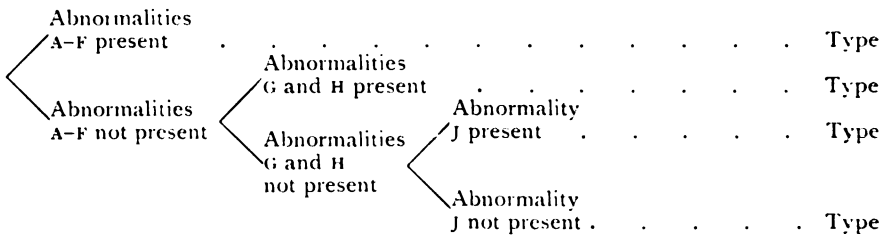
In this connection there is a possibility of error which should be clearly understood. It is simply that if mental deficiency or any other abnormality is turning up irregularly among children, then the more children a person has, the larger is his chance of having a mental defective. Consequently we would expect most defectives to come from large families simply because the families are bigger. This is clearly an important factor.

This complication can be avoided in two ways: either we can take the families as they come and apply a correction which will allow for this factor, or else we can collect our data so as to avoid the whole difficulty.

The mathematical form of the correction has been worked out and checked by two members of the committee. It is given in Appendix 2.

We have tested this question from several points of view.

Firstly the 415 families were sorted into four types according to the presence or absence of various conditions. The classification is shown in the following scheme:



In this way the families are classified roughly according to the seriousness of the abnormalities present. Thus Type 1 families include a person suffering from a major neurological or psychiatric disorder, while Type 4 families were entirely free from any recorded abnormalities.

The average size of family was then found for each type, and we examined the averages to see whether the more seriously affected families tended to be larger than the more normal. This, of course, is accompanied by a test for the statistical significance of such differences in mean size. On testing, the differences proved to be insignificant (Table II).

As the committee is more particularly interested in mental deficiency, we also divided the 415 families into two groups: (1) those containing a " weak-minded " person, and (2) those not containing such a person. We then compared the two average sizes of family. After correction for the error mentioned above, the difference was again insignificant (Tables III and IV). " Size of family " in the two tests above refers to all persons covered by the form.

In order to test a more sharply defined group, we have tested the number of sibs by dividing the 415 sibships into the two groups: (1) sibs all normal, and

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(2) sibs including one or more abnormal people. We then find the average size of the sibships in the two groups. Again after correction the difference was insignificant (Tables V and VI).

Finally, we have considered the question of whether parents marked as abnormal tend to have more children than the normal ones. This needs no correction, as the error mentioned above does not apply here. On testing, the difference is insignificant (Table VII).

This last test is particularly simple and to the point. We are taking parents who are marked as having some definite " neuropathic " disorder and we are recording the number of their children, and we are comparing this with the number of children from a very closely similar control group. It is difficult to imagine an experiment where the conditions would be more nearly ideal or more direct. Thus, since the youngest informant was aged 18 years, and the average age was 38 years, it is probable that most of the parents had completed their number of children. There seems to be no biased error which would affect the figures differentially, and the test is remarkably direct since we are examining precisely the reproduction of those with a " neuropathic " disorder.

There were 51 pairs of parents where one of them was abnormal and they had 273 children. The remaining 364 pairs who were both normal had 1,882 children. The average number of children for the two groups was 5·35 and 5·17 respectively. The difference is insignificant.

To check the point in more detail we have divided the abnormal parents into classes, e.g., those who were " tubercular ", " insane ", etc., and tested the average numbers of children in each class. The differences were again insignificant (Tables VIII and IX).

It may be objected that the numbers in the two groups are not very large. We may make the discussion more definite by using Fisher's method of "fiducial probability ". The difference between the two groups in our series is 0·18 children, i.e., the one group had, on the average, 0·18 more children than the other group. It may be shown that if there were a real difference between the two groups of more than 0·78 children, then our series would almost certainly have shown a significant difference. Our series, however, has given an insignificant difference. We conclude, therefore, that if there is any real difference between the two groups, it must be less than 0·78 children. This corresponds to 15%.

The fact that all five methods of approach have shown no significant difference between the abnormals and the normals is certainly interesting. The results are of extra value in that the records are taken very largely from one social class, so that the disturbing effects of social class are eliminated.

The committee is of the opinion that these results are not affected by any definite error other than the one allowed for, and they believe the results to be essentially reliable. The results seem to be conclusive in showing that

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there is no *gross* difference between the sizes of families in the two groups. Small differences, of course, could only be detected by the examination of very large numbers of families.

Finally, there were a number of other points on which the questionnaire might have given some information. But in these other points there were errors present which could neither be corrected nor measured. The committee feels that there is no justification for publishing data from which no reliable deductions can be drawn. These other points have therefore been omitted.

Summary.

A questionnaire was circulated in order to ascertain the incidence of a " neuropathic " history in the families of the mentally normal. This would naturally provide a basis for the discussion of the significance of the incidence of such histories in the families of the insane and the mentally defective.

415 forms were returned and examined.

(1) It was found that 57% of normal informants gave a " positive " family history. Excluding the minor conditions, 23% of informants gave a history of some major neurological or psychiatric abnormality in the family. It was known to the committee that certain errors were liable to be present, but all the errors would tend to give too low a figure. The committee are unanimously agreed that the figures given may be accepted as *lower limits* to the true figures. There does not appear to be any appreciable error which would make these figures too high.

(2) The forms also give some information on the question of whether the families which include abnormal persons tend to be larger than the more normal. Five different tests from various points of view all gave negative results.

(3) The necessary tables are given in Appendix 1.

Appendix 1.

Table I.

(For an explanation of the types, see the text.)

Type of family.	Number.	%	
Type 1	94	23	57%
„ 2	87	21	
„ 3	54	13	
„ 4	180	43	
Total	415	100	

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TABLE II.—*Average Size of Family in the Four Types of Family.*

	Total persons.	Number of families.	Average persons per family.
Type 1 families	2,350	94	25·0
„ 2 „	2,014	87	23·1
„ 3 „	1,361	54	25·2
„ 4 „	4,032	180	22·4
Total	9,757	415	

Test 1.—To test whether the differences in the four averages are significant. Using Fisher's z-test, $z = 0{\cdot}4974$, $n_1 = 3$, $n_2 = 411$, $P = 0{\cdot}05$. As the average for Type 4 families is the smallest, it is clear that correction can only render the differences quite insignificant.

TABLE III.—*Average Size of Family in the Two Groups: (1) Family includes a "Weak-minded" Person; (2) Family does not include such a Person.*

Type of family.	Number of people.	Number of families.	Average persons per family.
(1)	865	30	28·9
(2)	8,892	385	23·1
Total	9,757	415	

TABLE IV.—*Frequency of "Weak-mindedness".*

Type of person.	Number.
"Weak-minded"	37
Other abnormalities	578
Normal	9,142
Total	9,757

Test 2.—To test whether the difference between the two average sizes of family is significantly different from the difference expected according to Appendix 2.

Actual difference in family size . . = 5·737
Expected „ „ „ . . = 3·414

Deviation . . . = 2·323

$t = 1{\cdot}45$, $n = 413$, $P = 0{\cdot}15$.

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TABLE V.

This gives the distributions of the number of the informant's sibs (not counting the informant) in the two groups: (1) sibs all normal; (2) one or more sibs abnormal. One informant who marked himself as " tubercular " has been omitted.

Size of sibship.	(1)	(2)	Total.
1	44	1	45
2	52	9	61
3	51	12	63
4	33	15	48
5	40	15	55
6	33	10	43
7	18	10	28
8	14	8	22
9	11	3	14
10	7	1	8
11	3	2	5
12	—	—	—
13	1	1	2
Total	307	87	394*
Average sibs	4·18	5·24	

* The number is not 414 as some of the informants had no sibs.

TABLE VI.—*Occurrence of Abnormalities among the Sibs (including the Informants).*

Type of sib.	Number.
Abnormal	124
Normal	1,616
Total	1,740

Test 3.—To test whether the difference between the two average sizes of sibship is significantly different from the difference expected according to Appendix 2.

Actual difference between sizes of sibships . . =1·059
Expected „ „ „ „ . . =1·391

The actual difference is less than the expected difference, and therefore cannot be significantly greater.

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TABLE VII.

Distribution of number of sibs (including the informant) in the two groups: (1) Where both parents are normal; (2) where one or both parents are abnormal.

Size of sibship.	(1)	(2)	Total.
1	20	1	21
2	41	4	45
3	50	11	61
4	53	10	63
5	43	5	48
6	51	4	55
7	40	3	43
8	25	3	28
9	17	5	22
10	10	4	14
11	8	—	8
12	4	1	5
13	—	—	—
14	2	—	2
Total	364	51	415
Mean persons per sibship	5·17	5·35	

Test 4.—To test whether the mean of column (2) is significantly larger than the mean of column (1).

$$t = 0{\cdot}460,\ n = 413,\ P = 0{\cdot}65.$$

TABLE VIII.—*Distributions of Number of Sibs in Different Abnormalities of the Parent.* (*No case occurred where both parents were marked as abnormal.*)

Size of sibship.	Type of abnormality in parent. A	B	C	D	E	F	G	H	J	Total.
1	—	—	—	—	—	—	—	—	1	1
2	1	—	—	—	—	—	1	—	2	4
3	—	—	—	—	1	1	2	4	3	11
4	1	—	—	—	—	—	1	5	3	10
5	1	—	—	—	—	—	1	2	1	5
6	—	—	—	—	—	—	1	1	2	4
7	—	—	—	—	1	—	—	1	1	3
8	1	—	—	—	—	—	—	—	2	3
9	1	—	—	—	—	—	2	1	1	5
10	—	—	—	—	—	—	—	2	2	4
11	—	—	—	—	—	—	—	—	—	—
12	—	—	—	—	—	—	—	1	—	1
Total	5	—	—	—	2	1	8	17	18	51

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Test 5.—Do the different columns show significant differences in their means ?

TABLE IX.—*Analysis of Variance of Table VIII.*

Variance.	D of F.	Sum of sq.	Mean square.
Between means	5	8·0786	1·6157
Residual	45	363·5685	8·0793
Total	50	371·6471	—

As the mean square of the residual errors is the larger, the differences between the means must be insignificant.

Appendix 2.

If a given condition, e.g., mental deficiency, is occurring in families purely by chance, each child having an equal chance, p, of being mentally defective, then the larger the family the greater will be its chance of including a defective. Consequently, if we compare two groups of families, the first group having no mental defectives and the second group consisting of families each of which contains at least one defective, then the mean size (persons per family) of the latter group will tend to be larger.

It is required to estimate the difference between the two means and to devise a test of significance to compare the observed difference with the expected difference.

If we start with one family of n persons, the chance that none are defective is q^n, and the chance that it contains at least one affected person is $1-q^n$. So, of f_n families, each of n people, $f_n q^n$ families will go to Group 1 (above), while the remainder, $f_n (1-q^n)$ will go to Group 2. We may show the result best by giving a table of the expected frequencies (Table X) :

TABLE X.

Number of persons in family.	Number of families.	Group 1. Number of families.	Group 1. Number of persons.	Group 2. Number of families.	Group 2. Number of persons.
1	f_1	$f_1 q$	$f_1 q$	$f_1 (1-q)$	$f_1 (1-q)$
2	f_2	$f_2 q^2$	$2f_2 q^2$	$f_2 (1-q^2)$	$2f_2 (1-q^2)$
3	f_3	$f_3 q^3$	$3f_3 q^3$	$f_3 (1-q^3)$	$3f_3 (1-q^3)$
..	..	..	..	..	..
n	f_n	$f_n q^n$	$nf_n q^n$	$f_n (1-q^n)$	$nf_n (1-q^n)$
..	..	..	..	..	..

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Let the mean number of persons per family in Groups 1 and 2 be $\bar{x}_1$ and $\bar{x}_2$ respectively. Then—

$$\bar{x}_1 = \frac{\text{Number of persons}}{\text{Number of families}} = \frac{Snf_n q^n}{Sf_n q^n}$$

and similarly $\bar{x}_2 = \dfrac{Snf_n\,(1-q^n)}{Sf_n\,(1-q^n)}$.

From these we find $\bar{x}_2 - \bar{x}_1$. The values of f are given by the data. To estimate q we note that we have an unselected group of people, any of which may be mentally defective (after subtracting the informants who are necessarily normal). q will thus be given at once by—

$$q = 1 - \frac{\text{Number of defectives}}{\text{Number of possible defectives}}.$$

In calculating $Snf_n q^n$, etc., if each family contains one necessarily normal informant, we may allow for his presence simply by dropping one normal person from each family. We do not need to know which person the informant is.

The test of significance is straightforward, since " persons per family " is distributed in a form not grossly different from the normal form, so the observed means, $\bar{x}'_1$ and $\bar{x}'_2$, will be distributed practically normally. The variances of $\bar{x}'_1$ and $\bar{x}'_2$ are soon found. The difference, $\bar{x}'_2 - \bar{x}'_1$ will be distributed normally with variance equal to the sum of the two variances. So, by hypothesis, $\dfrac{(\bar{x}_2 - \bar{x}_1) - (\bar{x}'_2 - \bar{x}'_1)}{\sqrt{V}}$ will be distributed normally about zero with unit standard deviation. It may therefore be equated to t and Fisher's table used. No alteration in the degrees of freedom is needed, since q is calculated from other data.

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DIFFUSE WHITE MATTER GLIOSIS IN MENTAL DEFECTIVES.*

By A. MEYER, M.D.Bonn, and L. C. COOK, M.B.Camb., D.P.M.

(From the Central Pathological Laboratory of the London County Mental Hospitals.)

THIS paper is a preliminary report of some investigations we are now making into the pathology of mental deficiency, mainly from the histological point of view. We have so far examined 22 cases, comprising 7 low-grade defectives showing gross neurological lesions, chiefly of extrapyramidal character, 6 microcephalics with spastic diplegia and severe mental defect, 2 able-bodied microcephalics without gross neurological signs, 1 simple able-bodied idiot and 6 mongols. In this short paper it is impossible to give even the slightest indication of the wealth of pathological material found in such a variety of widely differing conditions. A full description of our findings, together with a more detailed discussion, must be left to further papers dealing with individual groups of cases. The purpose of making a short note at this stage is to draw attention to one particular finding which struck us by its surprising constancy, i.e., a proliferation of the fibrous glia, particularly pronounced within the cerebral and often also the cerebellar white matter. In many of the cases the cortical changes were slight compared with the intense lesions of the white matter. In none of them was demyelinization a well-marked feature, nor did its severity approach that of the glial proliferation ; in many it was negligible, and there were none of the fatty breakdown products, characteristic of the demyelinizing disease of the white matter called after Schilder. The gliosis was either diffused or patchy, and was often markedly perivascular ; even stripe-like scars, as described by Hallervorden (10), were to be seen, although, contrary to this author's observation, there was, as a rule, no corresponding myelin defect.

The gliosis of the white matter was observed in cases of *état dysmyélinisé* of the globus pallidus, *état marbré* of the striate body and in related progressive conditions, commencing at birth or in infancy. It was found in all but one of our microcephalics, whether they were associated with gross developmental disfiguration of the convolutional pattern or not. Its presence in " microcephalia vera " (8, 1, 12, 4, 14, 9), that is to say, in cases of pachygyria, microgyria and heterotopia not complicated by gross destructive lesions, such as

* Read at a Quarterly Meeting of the Royal Medico-Psychological Association, February 17, 1937. The detailed investigations on which this summary is based are embodied in a series of papers in course of publication in this journal.

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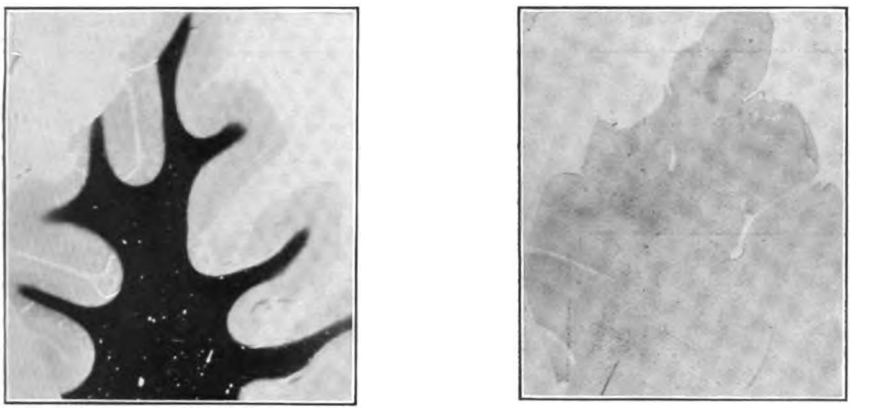
porencephaly or hydrocephaly, was quite unexpected. The maldeveloped cortex and the heterotopic areas within the white matter were always free from gliotic proliferation except for occasional sub-pial gliosis. The one exception mentioned above was a familial microcephalic. In this brain the most striking features were circumscribed heterotopias in the region between the insula and the putamen, and it must be more than a coincidence that H. Jacob (11) has described an exactly similar state of globular heterotopia without gliosis in familial microcephalics also. It may well be that cases of this type belong to a special group.

Of particular interest are naturally the findings in the brains of three able-bodied low-grade defectives and in all six mongols available for examination.



a Weil's myelin stain. *b* Holzer's glia stain.

FIG. 1.—Able-bodied defective.

One of the able-bodied microcephalics had, apart from the diffuse lesion in the white matter, slight scarring in the striate body and the thalamus. Another showed a mild demyelinization of the type seen in the Pelizæus-Merzbacher disease. In two of the mongols, incidentally the youngest in our material, who died at the age of 18 months and 3 years, the lesions within the white matter were very diffuse, whereas in the others they were of a patchy and perivascular type. Gliosis of longer standing was found combined with that of more recent appearance, somewhat independently of the age of the patients, which varied from 18 months to 20 years at the time of death. As a rule, subependymal regions were particularly affected.

A few photographs are included in order to show the characteristic localization of the gliosis. Fig. 1 shows a diffuse (though occasionally patchy) gliosis without demyelinization (Fig. 1*b*) in the white matter of one of the able-bodied microcephalics. Fig. 2 shows a similar condition in the white

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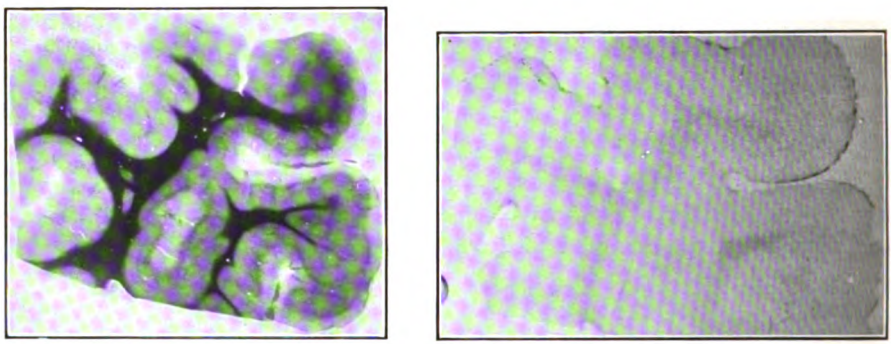
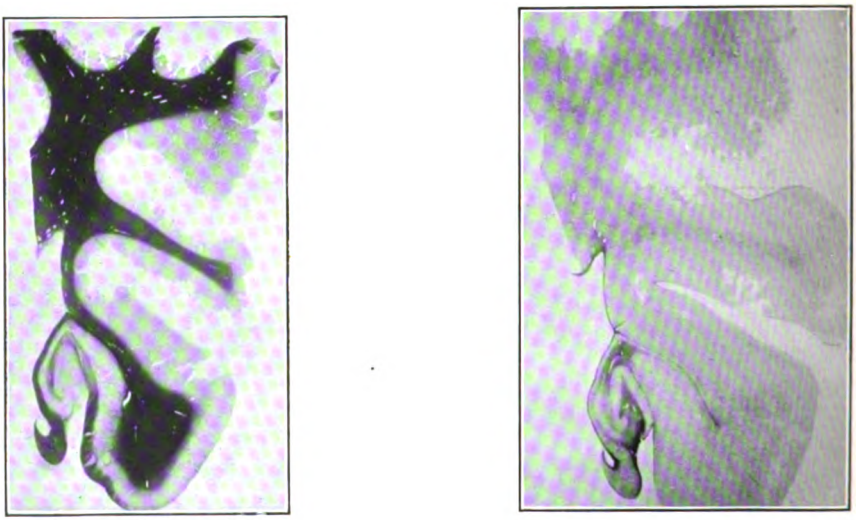
matter of a microgyric microcephalic. The clear spaces in the myelin picture (Fig. 2*b*) indicate heterotopic areas which are spared by the gliosis. The



a Myelin stain. *b* Glia stain.

FIG. 2.—Microgyria.



a Myelin stain. *b* Glia stain.

FIG. 3.—Mongol.

same characteristic white matter gliosis without a marked degree of demyelinization is shown in Fig. 3, which is taken from one of the younger mongols. The sclerosis of the vulnerable Sommer sector in the cornu ammonis should be especially noted. It will be seen from the photograph that the full extent of

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the lesions is brought out only by fibro-glial staining (Holzer), myelin and cell pictures giving no real indication of what we were to find. This perhaps explains why the glial scarring in these conditions has so far been largely overlooked, routine brain examinations and architectonic studies being often confined to cell and myelin stains.

Discussion.

An adequate attempt at interpreting the findings would require a far more detailed presentation of the clinical and pathological data than has been possible in this paper. We shall confine ourselves, therefore, to a brief discussion of the main points.

Gliosis, with little or no demyelinization and other breakdown changes, has been reported already by Freeman (7) in the brain-stem of diplegic microcephalics, and by Bodechtel and Guttmann (3) in cases of diffuse sclerosis. More recently Scherer (13) has described the same type of lesion in the cerebellar white matter as an important feature of olivo-ponto-cerebellar degeneration, and Davison (6) has found gliosis without demyelinization in the occipital lobes in Huntington's chorea. In all our cases the gliosis showed no difference from that seen in conditions of adult life, and there were none of the immature or blastomatous glia-cells characteristic of developmental errors, such as are to be seen in tuberous sclerosis, von Recklinghausen's disease, etc. The idea that non-developed brain-tissue is simply replaced by glia has little factual basis, and only serves to obscure the true pathogenic interpretation of the findings. As to the microgyric brains, the appearance of the white matter gliosis in regions where cortical disturbances were negligible, e.g., in the cerebellum, suggests that the gliosis was not secondary to the malformation of the cortex. It is interesting to note how often pathological processes accompany developmental lesions (in one case only of our series were developmental changes found alone, and this was significantly a familial microcephalic), and undoubtedly in some cases they may be their cause. More often than is realized developmental arrest is only the indicator of the time of the damage, and not the expression of an intrinsic, abiotrophic or faulty tendency of the tissue. This is a point of particular importance, because the belief that mental deficiency in general is due to an innate developmental inability, which might be diffuse or localized, is still widely popular in this country.

The pathogenesis of the gliosis is at present obscure. There is, however, in many of our cases some evidence suggesting that a vascular factor or some other type of defective tissue oxygenation might be the immediate cause of the change. The lesions peculiar to *état marbré* and *état dysmyélinisé* are considered by some to be due to vascular disturbances. In one of the mongols the Sommer sector of the cornu ammonis showed a well-marked sclerosis, and, in addition, there were, in the mongols and in other cases, perivascular

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changes as well as the diffuse lesions. The findings of Schwartz (15, 16) in birth-trauma and of Bodechtel (2) in congenital heart disease point in the same direction. It should be borne in mind, however, that defective tissue oxygenation is often merely a final common pathway of ætiologically different disease processes.

No generalizations can be made as to the glial changes acting as a substrate of the mental defect. This interpretation could be made only after evaluating all clinical and pathological data in the individual cases. Moreover, our material of able-bodied defectives, all three of them low grade, is too small to allow of generalization. The findings in these cases, however, give a forcible warning against indiscriminate use of such brains for comparative purposes, e.g., measurements of the cortex, which are liable to grave error unless a systematic investigation of the whole brain is made.

The interpretation of the pathological changes is particularly difficult in the mongols, who, apart from their mental defect, are distinguished by a feeble bodily constitution, frequently associated with disturbances of the vascular system and a susceptibility to infections of all kinds. The changes described, however, are unlikely to be due to the terminal disease, which was very short in our cases, death occurring even suddenly in some of them. Moreover, congenital heart disease was lacking in four of the six cases, including the case illustrated in Fig. 3. The constancy of the lesions is striking; they throw some new light on occasional observations reported by previous writers (see Davidoff's (5) review). Further evidence is required before the full significance of the changes can be assessed.

This necessary restraint in the interpretation of the findings should not obscure the fact that the extremely high incidence of lesions within the white matter adds a new feature to the pathology of mental deficiency. Bodechtel (2) has already expressed the view that it is the white matter which is particularly susceptible to oxygen deprivation in the developing brain, in contrast to the adult brain, in which the cortex is more vulnerable. Our own investigations confirm this on an unexpectedly large scale. In emphasizing the importance of the white matter, we do not wish to disparage that of the cortex, the integrity of which is, of course, essential to the development of normal intelligence. Traditional ideas, however, derived from genetic considerations, do make it necessary to stress the obvious conclusion that any research into the pathogenesis of mental defect requires impartial examination of all parts of the brain.

To Mrs. A. Meyer and Mr. Wright we owe thanks for the histological preparations, while for case-material and other help we are indebted to several colleagues, to whom individual acknowledgment will be made in subsequent papers.

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Discussion.

Dr. GUTTMANN expressed his appreciation of Prof. Meyer's interesting and impressive anatomical findings which he had just presented, especially his remarks on the able-bodied imbeciles whose brains he had examined. What Prof. Meyer had said naturally drew attention to the clinical side, and led one to ask what symptoms could one expect in patients with brains like those described and depicted ? There might be several reasons why so little was known about the symptoms in these so-called able-bodied imbeciles ; it might be that the symptoms were of a different kind from anything hitherto discovered ; or it might be they were not found because they were not looked for. Such findings as sclerosis of the grey matter of the occipital lobe pointed to the importance of investigating the visual functions. The main point seemed to be that there might be symptoms which were known, but their correlation with anatomical findings was not known. During the last decade a good deal had been learned about the anatomical basis of psychological symptoms. He reminded members of the disturbances of initiative when lesions were present in the frontal area of the brain, or disturbances of expression and of personality after encephalitis with its well-known anatomical lesions.

However, it was only to a small section of such symptoms and their possible anatomical relation that he wished to direct attention. Prof. Meyer had mentioned that perhaps the more exact analysis of disturbances which were known in imbeciles, such as their clumsiness, might lead to the possibility of correlating those symptoms with the anatomical findings.

He intended to show a few pictures ; he did not claim to show anything new. He asked members to look at those clinical pictures from a neurological angle, which need not exclude the psychological aspect of these conditions, but would simply give another approach to these problems. He was indebted to Dr. Lindsay, and especially to Dr. Earl, who gave him and his colleagues access to some of the material which would be seen in the film. This was only a sample of their collection showing things which everybody approached from the psychological side, but which were now being approached neurologically. The athetotic movements and facial expressions were now regarded as neurological symptoms and not as mere grimaces. It was difficult to determine these movements in stereotyped idiots. There were pictures of feeblemindedness which might point to sensory disturbance as being at the bottom of the whole clinical picture. Of that little was yet known ; they were only collecting information which possibly might be interpreted in the light of these anatomical findings.

[Dr. Guttmann here showed a film illustrating various disorders of motility.]

In the first part of the film there was shown a boy whose emotional expression started locally in the face, and then spread so that the whole body became violently contorted. Another boy shown had an athetotic lesion and a peculiar facial expression. Next there was a young woman who suffered from a disturbance of the first impulses in the act of walking. There was not yet a method of analysing them from the neurological standpoint. He directed attention to the patient's high stepping. There was also seen in the film a patient with motor infantilism ;

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it showed a method of grasping things without using the thumb; and there were movements similar to those seen in encephalitis.

Dr. W. R. ASHBY said that one point in Prof. Meyer's paper which had interested him very much was the widespread character of the lesions. That accorded with a view he had held for a long time, in contrast to statements in many of the earlier books. People who wrote on the subject years ago seemed to have the idea that intelligence was localized to some very small area of the brain. At first it was thought to be in the cortex, and later it was even more narrowly allocated to the frontal lobes, then to the prefrontal area. Many anatomists hoped, at that time, that they would ultimately find that small spot. His own view was that intelligence would be found to be more a function of the whole brain. The matter might be compared with the case of the athlete. No one expected the athlete to be the man with the longest possible legs, or the biggest heart; it was recognized that the athlete, to be first-class and efficient, must be a well-proportioned person. If his legs were made twice as long, then, instead of doubling his speed on the track, it would upset his performance. He asked whether Prof. Meyer considered that intelligence could be a function of the whole brain, and not one which was restricted to a particular area of it.

Dr. F. A. PICKWORTH said: The changes in the white matter of the cases which Prof. Meyer has described are often regarded as a primary gliosis, and I am glad to hear that Prof. Meyer believes that at least some are consequent upon an anoxæmia. Spasm or occlusion of blood-vessels is known to kill the vulnerable nerve-cells, allowing the more resistant glia-cells to survive, and these later increase with restoration of the blood-supply and the stimulus of the contiguous products of degeneration.

Taking mental deficiency in general I believe that occlusion of capillary brain vessels is a primary factor of real ætiological significance, and which is not necessarily restricted to any stage of development. In the developing embryo the period of competence of certain cells is strictly limited, and J. Needham showed that once this period has been passed without cell differentiation, no amount of auxins or sterols can ever make them develop normally afterwards. It is equally obvious that occlusion of nutrient vessels occurring at any period of intra-uterine life and in infancy hinders local growth, and results in a disproportionate development of such areas in respect to the rest of the brain. Environmental stresses necessitate that certain reflexes and mental activities shall be met by other functional systems to the detriment of the organism as a whole, and thus forming the essential basis of mental defect.

Of the causes of the vascular defects, birth trauma is probably the most important, although signs or symptoms of such injury may not be recognizable until long after birth. In my book on *Chronic Nasal Sinusitis and its Relation to Mental Disorder* I illustrate (fig. 26, p. 58) bacterial organisms as a cause of cerebral vascular defect. In this connection the importance of a bacteriæmia in the mother during the first few weeks of pregnancy is obvious, as recent researches have proved that a bacteriæmia from dental sepsis, for example, is of quite common occurrence. In the millions of chances provided by such bacteriæmia, an involvement of the placenta and passage of bacteria into the fœtal circulation to cause such changes in the cerebral capillaries is by no means a negligible consideration. A practitioner with much experience with mental defectives recently told me that he was very much impressed with the frequency he had obtained a history of a chronic septic condition in the mothers of defective children during the first few weeks of pregnancy.

An association of body deformities and metabolic or endocrine disturbances with mental deficiency is found in the pathetic creatures of institution nurseries, and such associations suggest vascular injury of the diencephalon. The vascular defects of spasm, thrombosis and hæmorrhage occurring at various stages of

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development and in various localities of the brain to my mind can account for many clinical syndromes at present considered hereditary diseases.

The mechanisms I have demonstrated in the adult, of the relation between nasal sinus infection and pituitary hypothalamic dysfunction, are of equal importance in infancy, since chronic nasal infection may be established within a few weeks of birth. The specimen now shown is the sphenoid of a child only three months old, but which contains pus in the left spheno-ethmoidal recess as the most striking post-mortem finding. As a student I saw a case of mental deficiency which developed normally until nine months old, when a discharge from the ear was noted (the ear condition being probably part of a general nasal sinus infection), and only many months after this infection had subsided did the absence of further mental development become apparent. The clinico-pathological picture is that of a subclinical basal meningitis with thromboses of many of the cerebral capillaries to such an extent that further local cerebral development was inhibited until too late. Such cases, I believe, are commoner than generally supposed, and a recognition of this type of ætiological pathology will bring many cases of incipient mental defect within the realm of economic and preventive medicine.

Dr. JONES (Caterham) said he would like to tell the meeting about a case which he examined after death last month, and which supported Prof. Meyer's contention. He was an able-bodied imbecile, and the white matter of the brain showed dense gliosis in the occipital region, in the cerebellum and in the brain-stem. The characteristic was that the corresponding myelin picture showed either normal demyelinization or only a small fraction of the normal. The gliosis was most marked in the occipital region, and in the islands of the cerebellum and the brain-stem. There was a rarefaction in the centre of the white matter. He showed a slide demonstrating this case, and another slide showing corresponding gliosis in the region of the fibres, without demyelinization.

Dr. W. F. MENZIES said he wished, first, to correct a false impression. Dr. Ashby had just said that intellectual power had been supposed to be resident in one small part of the cortex. He, the speaker, had never heard a psychiatrist put forward such a view. For fifty years it had been known that intelligence was a matter of association of every part of the brain.

He wished to mention two cases of choreo-athetosis which were in the imbecile block at Cheddleton, in respect of which he would like some information. Why had those cases such extreme hypogonadism? The brain of one was, when he left Cheddleton, in process of being hardened. That boy could not speak at all, and when he tried to he made violent contortions, and these were seen whenever he tried to express any emotion, the motions affecting the whole body. The bowel and bladder reflexes, however, were not interfered with at all, and apparently his intelligence was normal when judged by his replies to questions; he enjoyed the cinema and liked to watch dancing. He had extreme hypogonadism; up to eighteen years of age he had no hair on the pubes, and the penis and testicles were very small. Then suddenly, in the course of the next year, an immense growth of the generative organs took place. Shortly afterwards he caught influenza, then pneumonia, and died.

The other case was that of a girl, now about twenty years of age, and very intelligent, in fact, a nice child. She used to like the nurse to read stories to her, and she especially enjoyed fairy tales, which she seemed to understand. But she was unable to speak a word. She was now learning to say a very little, and responded to questions with the ejaculation "Ya" if she approved, shaking her head in token of negation. She, also, had extreme hypogonadism; she had never menstruated, had no pubic or axillary hair, and the mammæ were not developed. Her glands were almost intangible. He asked whether those who had investigated cases of choreo-athetosis had gone into the question of the gonads.

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Dr. B. H. SHAW remarked that the cases which had been described and depicted were those with very grave defects. He had noticed pathologically that in cases of mild mental deficiency there was a definite lessening of the granular nuclei in the cerebellum. Normally the diameter was a very constant figure—4·5μ—but in mental enfeeblement the diameter was much less—as much as 25% less, for in many of these defectives it was no more than 3·7. That he regarded as of some moment.

With regard to the question of gliosis, he had had two cases among his epileptics with peculiar movements. Both cases, when they had a fit, revolved in the same direction, i.e., right to left, quickly, just before the actual fit, and continued afterwards. In both cases he found that the lesion was a gliosis of the periphery of the cerebellar cortex on the right side. The fits dated from an attack of acute rheumatism.

Dr. MEYER, in reply, thanked the members very much for their kind reception of this crude attempt to tackle again, after so many failures, the big problem of mental deficiency. He was particularly indebted to Drs. Earl and Guttmann, as they had rendered him most valuable services, especially in putting his description into a more clinical form. It was that which he had in mind when he asked for a more refined neurological approach in regard to the so-called able-bodied defectives.

In reply to Dr. Ashby, he would consider that it was always the whole brain which worked in normal and in pathological conditions; but there might be a localizable defect, which might differ in site with different conditions. It was possible that future analyses of mental defectives would make it possible to differentiate between the one whose visual perceptive side was good, and the one who had a number of defects. Earl had pointed out that it was the emotional sphere which had hindered normal development, and it was that which might even help, under certain circumstances, to overcome the defect.

What Dr. Ashby did not mention was another interesting point brought about by his own research on the biochemical side. It had been pointed out that some biochemical abnormality might be due to lack of neurons, or to an overgrowth of neuroglia, and he would like an opinion as to whether that overgrowth of neuroglia in able-bodied people did not fit in with some of Dr. Ashby's conceptions.

He agreed with what Dr. Pickworth said. He was sorry he had been misunderstood. Nothing could be known about the nature of the process unless one knew the earlier stages; and from what he had seen he stressed the possibility of anoxæmia being a possible cause. That would fit in with Dr. Pickworth's suggestions and findings.

He thanked Dr. Jones for his remarks, because that gentleman brought the first confirmation of the gliosis, which now enlarged the very limited material, and consolidated a little the position which had been taken up.

With regard to the remarks of Dr. Menzies on hypogonadism, in his own cases of choreo-athetosis there were no lesions of the testicles found, but choreo-athetosis was a symptom only. The choreo-athetotic cases he had shown were probably due to birth trauma and to anoxæmia. He had seen many cases of choreo-athetosis which would not show the changes he had described. He had seen a case of amaurotic idiocy developing from the choreo-athetotic stage and passing into a demented stage. Without examination of the brains of the cases of which Dr. Menzies spoke it was impossible to form a definite opinion on hypogonadism. It was, however, very important to keep this in mind, he thought, and not to omit examination of the bodily organs, not only the testicles, but also the pituitary and other endocrine glands.

He was sorry not to have heard any suggestions in regard to the findings in mongols, which were so characteristic of anoxæmia. He had had many ideas about it; and one thing which was worth discussing was that heredity in mongolism was no longer recognized as a factor. According to Penrose and others it seemed to be the advanced age of the mother which was one of the most important factors; it might be responsible for an insufficient blood-supply reaching and nourishing

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the fœtus—the kind of thing which Dr. Pickworth probably had in mind. More facts, however, were needed to enable a sound opinion to be formed.

With regard to Dr. Shaw's remark concerning the granular layer in high-grade defectives, as he, the speaker, stated in his paper, he had not seen high-grade defectives, therefore he could not reply on that point. It was a very interesting one, and deserved full attention. If there was a deficient granular layer, one must consider whether there was or was not something behind that. He could well imagine that an affection of the white matter would also possibly lead to such a diminution of the granular layer. In his own severe case the granular layer was intact; it was only the white matter in the hilum of the dentate nucleus which was severely affected.

Again he thanked his hearers for their patient hearing.

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ASPECTS OF TEMPERAMENT IN ADOLESCENT MALE OFFENDERS.*

By H. T. P. YOUNG, M.B., Ch.B.Ed.,
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A CONSIDERABLE diversity of opinion exists on the definition of that part of the personality which is known as temperament, and some authorities go so far as to say that no practical advantage is to be obtained in differentiating it from character. Temperament, nevertheless, is the means in every-day use by which strangers provisionally gauge the extent of their affinity to each other.

The nature of it is a matter which has lent itself to philosophical abstraction from remote times, and while its consideration by historians, theologians, phrenologists, biometricians, anatomists, physiologists and others has resulted in a variety of conceptions, these lines of approach have all tended to converge on a central point of common agreement. In doing so they have paved a way which should ultimately lead to a clearer understanding of the subject than would otherwise have been possible.

Any attempt to recapitulate the work of the various schools would much exceed the scope of this paper, but it is possible to outline broadly the developments leading up to present-day views. The temperaments described by Aristotle were based on the Hippocratic humoral doctrine of four types—those of the blood, the phlegm, the yellow and the black bile. These were again classified by Galen, and after various vicissitudes were reaffirmed by Kant under the following designations: the sanguine, phlegmatic, choleric and melancholic, the first and last of these being associated more directly with feeling and the others with the emotions.

Temperamental variations were formerly ascribed to the condition of the blood—Aristotle adducing its coagulability as the cause, and Kant attributing them to its flow and temperature.

The terms used by Kant have a descriptive value which many other classifications lack. Thus the sanguine temperament, as its name implies, signifies a careless, light-hearted type of pronounced but gradually decreasing determining capacity; the phlegmatic represents those who show reduced excitability and motivation, coupled with a strong determining disposition

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which slowly diminishes in intensity; the choleric indicates the type of increased motor and sensory excitability, with a weak determining disposition, requiring a greater effort to bring an act to fruition; and the melancholic describes the individual of weak determination and diminished sensory and motor excitability, resulting in a general ineffectiveness.

Although there is ample historical evidence of the consideration which the subject has received in books and learned dissertations from medieval times onwards, it is not proposed to do more than allude to the fact here. The work of Lavater on physiognomy in the eighteenth century may, however, be mentioned, for although he advocated the assessment of temperament in the "thermometric" manner, thus suggesting a connection between it and the body temperature, he considered it impossible to classify the various types by the preponderating chemical ingredient of the blood, since the effect on the bodily systems and appearance of certain salts, even in minimal amounts, could not be ignored. The phrenologists Gall and Spurzheim also introduced fresh conceptions in their correlation of mental and psychological characteristics with the physiognomical configuration, and Laycock, followed by Paget, laid stress on its periodical aspect. The latter emphasized the view that "each organ has its time rate of rise and decline in activity", instancing cases of families in which there was a rapid attainment of maturity followed by a slow decline, and the reverse of this process in others.

In recent times Sigaud established four types described as the respiratory, the digestive, the muscular and the cerebral, in accordance with their reaction to atmospheric, alimentary, physical and social stimuli.

The number and variety of methods of estimating temperament, of which only a few instances have been given, testify, therefore, to the incompleteness and general inapplicability of any one classification, but the fact that common usage still adheres to the original nomenclature in describing the different varieties is of special interest, in view of the trend of modern biochemical research, particularly that in connection with the chemical agencies at the synapses. From these neurological studies it has been inferred that temperament and the endocrine gland system are closely linked.

Thus, Kretschmer, combining the anthropological and hormonic features of his cases, has correlated his two principal temperaments, the schizothyme and the cyclothyme, with physical characteristics classified under four groups—the asthenic, athletic, pyknic and hypoplastic. Even these types cannot be regarded as capable of fulfilling every requirement, for in adolescents particularly it may be difficult to determine the physical group in which an individual should be placed, and it requires a courageous diagnostician to decide to which of the temperamental types some cases belong, or, indeed, if a case of early mental disorder is undoubtedly schizophrenic or manic-depressive, for it is not very unusual to find symptoms compatible with either in the same person.

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It is generally accepted, however, that temperament is a characteristic feature of the personality resulting from the interaction of two diatheses, the physical and the psychical, and linking affection and conation with the deeper physiological levels. Its effect is regulated by the physical state and mental disposition of the individual, and emerges when thought produces, or tends to produce, deliberate or impulsive action. Temperamental trends have a prevailing direction in each case, and while they are inborn, Osler emphasizes the modifiability of temperamental tone by voluntary effort, and McDougall draws attention to its susceptibility to environmental influences. There is also evidence to show that it is remittent in its action.

Heterogeneous as temperamental types are, it is the pattern only which is peculiar to the individual, for the psychical and physical dispositions from which it arises are common in different degrees to all persons. A pattern, as, for example, in a lattice, is formed by the intersection of two series of parallel lines, and to depict temperament by this means it is necessary that both psychical and physical diatheses should be represented. That attempts were inadequate to classify it by the degree of subjectiveness or objectiveness present or by means of the introversion-extraversion scale, independently, was recognized by Dr. Beatrice Hinkle, who employed both criteria as a means of evaluation.

In the writer's view, both subjectiveness (which McDougall regards as closely related to a strong emotional constitution) and objectiveness (which is allied to a weak one), and also the introversion–extraversion scale, approach too closely to one aspect of temperament to comply with the metaphorical pattern if they are used in conjunction, and it is suggested that a more characteristic one may be envisaged by the employment of one of these scales, such as Jung's, and another criterion more closely related to the physiological aspect of temperament. A hypothetical design formed in this way cannot be set down in diagrammatic form, but this is not considered disadvantageous as the diathesis it represents is not amenable to this method of illustration.

Before dealing with it, however, a brief reference may be made to the acceleration and retardation of mental processes brought about by psychophysical interaction. The former is characteristic of the extravert in whom resistance is reduced at the point of transition from thought into action, while the latter is a feature of the introvert who has difficulty in overcoming this resistance. Extraversion and introversion, however, are not necesarily permanent states, for their fixity varies from time to time in the life of the individual, and while the extravert aspect is involved in the relationship between the self and other individuals, the introvert tendency is concerned with the subjective aspect only. The measure of restraint in the expression of those emotions and impulses provoked in any situation diminishes with the degree of extraversion present, while the introvert conceals himself behind a screen of reflection and brooding and thus avoids their overt expression. Absolute

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introversion and extraversion are the extremities of a scale, the features of which become less distinct as they approach the middle zone.

Recent work in America has shown that offenders, particularly those of the recidivist type, tend to converge towards the middle of the scale, but this observation does not necessarily differentiate them from the unconvicted classes of similar age and status, although these are stated to show more marked extravertive or introvertive traits. Indeed, the fact of imprisonment may be a cause of weighting the scale in favour of introversion owing to certain influences to which it is proposed to allude subsequently.

In an attempt to classify 1,000 young offenders between the ages of 16 and 21 years by means of this scale, figures have been obtained at Wormwood Scrubs which can only be advanced with considerable caution. It was found that out of this number 130 might be classed as extraverts, 344 as introverts, the remaining 526 being placed at some indefinite point between them. The grouping of these cases bears a superficial resemblance to the observations already referred to, but while a number of them may have committed several previous offences, none can be classed as recidivists in the usually accepted sense, for relatively few had experienced previous terms of imprisonment, and recidivism implies a habitual relapse into crime which occurs at a more mature age.

It will be seen that as the sole means of assessing temperamental qualities this classification is of limited value, but it is suggested that this does not justify the rejection of temperament as a separate entity of practical utility. Workers in vocational selection have given considerable attention to the matter in conjunction with the mental, intelligential and physical aspects of their cases, but it would seem that they approached it by estimating such qualities as ambition, sociability, cheerfulness, complacency, perseverance, energy and others, thus combining the features of temperament with those of character.

Reference has been made to another phase which has been considered in the past, but which on the whole has received less attention than it deserves. I allude to the periodic aspect of temperament, which appears to have given way to more materialistic psychological views, but the latter, nevertheless, do not afford an adequate explanation of the fact that mechanisms formed to deal with specific situations are not invariably so used. It is therefore proposed to examine this point in some detail in connection with temperament.

The derivation of the word "temperament" indicates the association between time and measurement. This division or apportionment in the time factor may be exhibited in many ways, and the strength or weakness of the responses to similar conditions may be observed to vary in the same individual at different times. Certain external agencies also are liable to a similar periodical movement.

Thomas Laycock in 1840 summarized the views held by some of those of

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his time in a *Treatise on Nervous Diseases in Women*, in which, after referring to the periodic movement in seasonal, astronomical, atmospherical, tidal and other phenomena, he states that " not only are there periodic influences ever-acting upon man, which are derived from without, but from the moment of conception (and perhaps before) series of changes commence, enchained by definite laws and depending for their due development upon agencies seated in the individual organism ". He then deals with the menstrual cycle, gestation and the chronological sequence of embryonal development, the septennial phases of life, puberty, the climacterium, sleeping and waking, the respiratory and cardiac rhythms and recurring functions and appetites, and after alluding to the periodic character of certain nervous disorders he says, " every vital act is more easily performed the oftener it is repeated "—a statement which needs no emphasis.

The disturbance of the rhythm of many of those " tendencies seated in the individual organism " results in overt interference with health. Periodicity in certain extraneous phenomena, for example that of night and day, can be readily appreciated, but the more indistinct the variations become the less readily can mental and physical reactions be correlated with them until the causes themselves cease to be recognizable and only their effects can be registered.

An instance of glandular insufficiency may be quoted to illustrate the effect on temperament of an organic disorder which was unappreciated by the subject as it had no adverse influence on his health, but was nevertheless directly concerned with his delinquency as a result of its effect upon his development :

C. P. S—, æt. 17 years 9 months. Convicted of stealing from churches. Sentenced to three years' Borstal detention.

This lad was in fair physical health, but showed extremely retarded physical development, having only just attained puberty. His physique was equivalent to that of a boy of fourteen.

His intelligence was fully up to the normal for his age. There was no evidence of psychoneurosis or psychosis, and he refused to accept any suggestion that he was not normal in every way.

He could give no explanation of his thefts, although difficulties at home with his father and stepmother might have been contributory causes. He was described as impudent, plausible, irritable and apt to threaten his brother —all being traits which would be associated with an intelligent lad who was physically retarded and labouring under the stress of a delayed adolescence. These, together with his delinquency, also suggested compensatory behaviour from a feeling of physical inadequacy in an extraverted personality.

The following case exemplifies a psychopathic or neuropathic reaction of a delinquent type to a severe physical trauma :

M. H. P—, æt. 19. Sentenced to three years' Borstal detention for theft.

The father was dead, the mother alive. An uncle died in a mental hospital

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and an aunt died of tuberculosis. One brother had fainting fits. There was no other morbid family history.

The lad had good but indulgent home surroundings. He was educated at a secondary school, where he was frequently top of his class and was captain of cricket and football. He left there at 15½ years of age.

He was somewhat reserved in manner. He studied Russian, Chinese, Spanish, German and the Indian Vedas, and was interested in foreign customs and religions.

His sex life was not abnormal, and he had a girl friend for a short period.

On leaving school he joined the Army and got on well. After eight months' service he passed an examination for entrance to Sandhurst. A week later he fell in the gymnasium and injured his spine. He was detained in a military hospital for four months, when he was invalided out of the Army with scoliosis. He became depressed at the loss of his career and developed attacks of pain and faintness, for which he underwent a manipulative operation at a large London hospital. He then developed a desire to inflict injuries on himself by cutting, which afforded him relief and satisfaction. This desire was not apparently of masochistic origin, and he denied feelings of guilt or unworthiness. He was treated for this for a considerable time by a psychological specialist at the same hospital, and was attending there as an out-patient at the time of arrest.

Some improvement took place at first and he went to work, but after five months he felt physically incapable of going on with it. After six months' idleness he began work in an office, but gave this up on account of spinal pain. He then remained out of work until his arrest.

He apparently appreciated the fact that his symptoms were not entirely due to organic causes, for he read books by Jung, Freud and Adler, but he could find no case analogous to his own. He slept badly, and was constantly labouring under a feeling of frustration and apprehensiveness about his spine. He also stated that he had a short period of amnesia, possibly a fugue, twelve months before arrest.

He was then bound over for stealing jewellery from a friend with whom he was staying, and during his probation he stole 3*s*. from another friend. For this offence he received his present sentence. He stated that he was not in need of money on either occasion.

While under observation he inflicted numerous wounds with various sharp objects on the right forearm on several occasions. The wounds were multiple and at times were severe. He interfered with his dressings occasionally, and once applied his bandage as a tourniquet, causing swelling of the hand. He also burnt his right wrist with a hot dinner-plate. Advice, warning and restraint had no beneficial effect. He insisted that the attacks were compulsive, and although he knew when they were coming on by preliminary head pains on the left side, he could not always restrain himself, although he often rang his bell when he felt an attack impending. He said that the injuries caused him relief, and that if he resisted the impulses he felt ill and depressed. This explanation, however, was of doubtful value.

The lad was depressed, introspective and introverted. His Columbian group test was 90%.

The frequency of the attacks has shown a marked diminution at the Borstal institution to which he was transferred. Apart from slight scoliosis, which was not considered sufficient to cause the symptoms he described, his physical condition was good.

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The case of C. W— illustrates an introverted reaction to a known psychic trauma :

He was æt. 18 years 11 months. Sentenced to three years' Borstal detention for begging and breach of probation.

His mother died during his childhood. The father and stepmother with three older siblings and one half-brother were living. There was no known morbid family history.

The lad gave a history of fainting attacks, regarded in the Army as epileptic, but observation showed no evidence of epilepsy, insanity or mental deficiency. The Columbian group test was 77%.

Scars of old osteomyelitis of the leg were present, but there was no sign of organic disease. He was dull and morose in manner and his conversation was whining. There was also evidence of sulkiness, bad temper and antisocial habits, indicating a lad of markedly introverted propensities. He was devoid of interest in his surroundings, and lacked initiative. This reaction was regarded as attributable to his environmental difficulties.

He felt himself to be unwanted by his parents from the time of his father's second marriage, and he stated that he was harshly treated by his stepmother, who alienated his father's affections. This was corroborated by inquiries made on behalf of the nautical school he subsequently attended.

The lad had been out of touch with his parents for three years before arrest. His delinquent history began at the age of 11, when he stole a bicycle to assist him in delivering papers, and when it in turn was stolen from him he ran away from home to avoid punishment for losing his work, and was eventually arrested for being without visible means of support.

He was sent to the nautical school until 15½ years of age, when he joined the Army, being discharged after three months because of a seizure. He again wandered from home, and left his next job one month later, after stealing £3, because his wages would not support him. On his return from a trip at sea he was charged with this, and returned to the nautical school. He was later bound over for two years for failing to carry out the terms of his licence.

After re-enlisting in the Army and committing further delinquencies he received the present sentence. He stated that he felt relieved at this, and it was anticipated that his affective life would revive at Borstal. After-care was regarded as being as important as training.

The following case is an example of a reaction to an adverse external influence, which was unidentified by the subject but which led nevertheless to persistent delinquency :

B. C. F—, æt. 19. Sentenced to three years' Borstal detention for breaking and entering.

This lad came under observation in consequence of his perverse conduct at a Borstal institution.

He was the illegitimate child of a woman, æt. 20, of good social status, and an Army officer who had held administrative posts in the colonies and was killed in the war. There is no known history of antisocial conduct or other abnormality on the part of either parent. The lad had no recollection of his mother, and knew of no other relatives. The mother's subsequent history is unknown.

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He was brought up in an orphanage until the age of two, when he was adopted by a respectable couple in prosperous circumstances, the adoptive father being a manager of a big stores in the provinces. The only son had died, but there were two older daughters living. The home had, it appeared, a rather Victorian atmosphere. The father was a man of strong convictions, and required a satisfactory reason for every request made by the boy. His methodical habits were exemplified in the way he preserved and docketed all letters from and about the boy. This attitude may have led to repressions in the lad, although he was fond of his home and was happy with his adoptive mother and one of the daughters.

Until the age of 15 he was ignorant of the fact that he was not a member of the family and was of illegitimate parentage, although both his adoptive sisters knew of it.

While at home he was a source of anxiety from an early age owing to his difficult and uncertain conduct. He attended day schools until the age of 10, when he went as a boarder in the preparatory school attached to one of the lesser public schools, where he remained for four years. His conduct there immediately gave rise to adverse comment. He ran away, broke rules, stole money from other boys, neglected his work and was frequently punished, without effect. He was moved into the senior school at the age of 14, where his irregular conduct continued. He was a failure at games, and he did not apply himself to his work. In spite of this he stated that he liked his house, the boys and the masters. He admitted that he gave no thought to the consequences of his acts. He interested himself in a superficial way in Egyptology and other unusual subjects, but persevered in none of them, and had no ambition of any kind. He committed numerous petty offences, on which punishment, threats or kindness had no effect. He occasionally practised masturbation with heterosexual fantasies, but not as a result of any serious sex trauma. In consequence of his unsatisfactory conduct his adoptive father told him, at the age of 15, of his parentage, and from that time the lad has had the knowledge of his illegitimacy constantly in mind, although he is not known to have discussed it spontaneously. He assumed his correct name when he left school. At the age of 16 he was removed, at the headmaster's request, for stealing. The lad was sorry to go. He was then placed as an apprentice in a department of his adoptive father's store, and attended night classes at a technical school. He did not like this, as he felt that he should have been given a superior post. His conduct again became unsatisfactory. He sold his bicycle and said that it had been stolen, he failed to attend to his work or classes, and, after six months, as he appeared to be quite unfitted for the business and showed an interest in flying, he was apprenticed to an aeroplane engineering firm of good standing. While there he kept the fees which he should have paid for his technical classes and spent them on cinemas, and he evaded his work. He also associated with a married woman and had sexual intercourse with her and other women. After taking away motor cars without leave on several occasions to entertain women friends, he was eventually charged with stealing a car and was bound over. His apprenticeship was thereupon cancelled.

It was then decided that he should prepare for matriculation, but this was abandoned when a friend of his adoptive father offered to take him at a premium into his engineering business with prospects of an interest in the firm. Towards the end of the first six months his feckless conduct returned. He pried into private cupboards and made trouble among other employees.

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He also made a childish attempt to hold up a shop assistant with a toy revolver because he needed money. He was, therefore, discharged.

His adoptive father then told him he could do no more for him and threw him on his own resources. He first sold articles from door to door, and then obtained employment in a shop for a week, when he stole £2 and absconded. The Labour Exchange then found him seasonal employment at a seaside resort, but he was paid off after a fortnight. He drove a tradesman's van for three weeks at a wage of 40s. a week, and while so employed he committed the offence, in association with another lad, for which he received his Borstal sentence. He was not in debt at the time, although he had frequently received money from his father to pay his debts in the past.

At the Borstal institution he soon became restless and eventually stole a car and went home. On examination the lad denied that he had been reproached about his illegitimacy at any time. He admitted that he had always successfully relied on his adoptive father, employer, landlady or friends to get him out of his difficulties. He had no plans for the future, as he anticipated that his adoptive parents would come to his assistance on release. He was of a somewhat effeminate type, suggestible, self-indulgent and lacking in foresight; his sentiment development and instinctive drive were poor.

His general health was good. He had had an operation for appendicitis at the age of 10, but had had no other serious illnesses or accidents. Apart from his defective vision his physical condition was sound. He denied any addiction to alcohol, but admitted that he was a heavy smoker. There were no signs of epilepsy or insanity. He was an extraverted type of superior intelligence (Columbian group test 88%) and was not lacking in general capacity. There was no evidence of mental or moral deficiency.

The lad's subsequent conduct has shown little change. Although his ability and intelligence are regarded as considerably above normal, he has not made use of them in his work, and has failed to progress. He is involved in any insubordinate conduct that may take place, he has made foolish attempts to abscond, and he will not undertake to abstain from further delinquency on release unless his adoptive father continues to help him.

This case is recorded in detail for the following reasons:

(1) The lad's misconduct began many years before he learned of his illegitimacy and adoption.

(2) His adopted sisters, who were æt. 9 and 10 respectively at the time he entered their home, were consequently aware that he was unrelated to the family and also probably that a veil was drawn over his antecedents, and they could not have been expected to be able to disguise this knowledge from the lad entirely.

(3) A child may not be able to avoid a feeling of alienation from his surroundings when he is brought up by people other than his own parents, in spite of their apparently kindly disposition towards him. This feeling of alienation is likely to be increased by the stigma of illegitimacy, however carefully it is intended to conceal it, though it is not of serious consequence to the children of irregular unions who have a parental upbringing. It is, however, comparable with that which is found from time to time in the children of marriages which are grossly incompatible, either in status, religion

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or race. In all of these the disparity produces a state of uncertainty in the mind of the offspring which may show itself in capricious or perverse conduct in childhood, and in antisocial reactions such as prostitution or licentiousness in later life.

(4) It is impossible to avoid the presumption of inherited abnormality in this case, though it is by no means inevitable that inherited abnormality necessarily implies later delinquency. Indeed, it is recognized that the hereditary factor is not so essential to the manifestation of antisocial acts as some writers would suggest—for example, Lange in his investigation of twins—for heredity cannot be held responsible for the implantation of any specific tendency to general delinquency.

In the present case no family history is available, but it is known that the adoptive father, who could be relied upon to investigate such a history thoroughly as he clearly intended the boy to take his son's place, and brought him up accordingly, had some personal knowledge of the lad's parents, which did not, in fact, deter him from carrying out his plan.

(5) The case is regarded as an example of aberrant personality type with defective sentiment formation, whose deviation from the normal was probably much increased by his temperamental constitution and circumstances which were unfavourable to him.

It also illustrates a further interpretation of temperament to which a brief allusion may be made.

Lewy, in his classification of types into the skilled, the clumsy, the asthenic and the tetanoid, adduced the differences in "fundamental tempo" in individuals as a cause of variance between them. It is thought that it may also explain some of the difficulties which adolescents may experience when removed from one kind of environment to an institution largely composed of lads drawn from another.

Fouillée also referred to the "rhythm of nature" exemplified in the alternating processes of re-integration and disintegration, and he deduced therefrom his saving and spending temperaments. In accordance with these two connotations the words "rhythm" and "periodicity" may be employed in a wide and comprehensive sense, and I propose at this point to consider them in greater detail.

The list of states in which periodical variations occur may, of course, be widely extended to include conditions as diverse as dipsomania and the drug addictions, the phases in the evolution of the sex and other instincts, fatigability and the alternations observed in nervous and mental states, such as the compulsive-obsessive psychoneurosis and the manic-depressive psychosis among others.

As an example of the effect of periodicity, imposed from without, the diminution in the frequency of fits in cases of epilepsy undergoing institutional care may be cited.

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Again the twin subjects of epidemiology and immunity are interesting in this connection, for in certain infections the former shows periodicity on the part of the invading parasite, and the latter on that of the host. The coincidence of increased virulence on the one hand and increased susceptibility on the other results in a general way in a successful synchronization, the effect of which may follow a definite periodic course. Malaria and encephalitis lethargica may be quoted as examples of different types.

There is little difficulty in recalling examples of this kind in which more or less specific rhythmic responses at physiological levels are induced by known agents, but in rhythmic processes of other types, such as are seen in the migration of birds, the cause is not always clear, for in certain species this has no apparent connection with the reproductive or self-preservative instincts. The upward fluctuations in the frequency of certain offences, attempted suicide, for example, occur at certain times and seasons and are generally associated with known predisposing causes, but the sharp rises in the total crime rate, although due principally to an increase in acquisitive offences, are not necessarily the result of poverty or prolonged unemployment, nor can the fall in rate in time of war be ascribed solely to the increased employment available. It is a significant fact that destitution among adolescent offenders is responsible for no more than 6·5% of the acquisitive offences committed by this class of delinquent, among whom crimes of acquisition account for 96% of the total. In another series of 500 consecutive cases 231 or 46·2% were unemployed and 269 or 53·8% were employed at the time of arrest. Of the total who were unemployed at that time, 33·4% committed offences within one month of losing their work, 16·0% within two months, the remaining 50·6% having been unemployed for more protracted periods.

The phrase "fundamental tempo" which has been mentioned previously is as liable to misinterpretation as are these external agencies, but it is sometimes to be observed when comparing rural and urban types. In the former there is frequently a retardation in psychomotility which is not invariably associated with differences in intelligence. This is reflected in the bodily movements, which are apt to be slower, and in the mental processes, in which there is a less rapid response to stimuli. The intolerance of town life in the one case and of country life in the other appears to be partly due to the difference in periodicity between the individual and his environment.

In this connection it is interesting to note that the migration of young workers from the rural districts of Japan towards the highly organized factory life of the towns is followed by an almost equally large movement of similar migrants in the reverse direction, suggesting that although rural workers are prepared to undergo for a certain time a routine which is highly systematized, they are unwilling, even at a very considerable sacrifice in their earnings, to accommodate themselves indefinitely to the exacting rhythm of industrial life.

Again, there are certain rare types whose rhythm synchronizes best with

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that which exists in states of serious emergency, and therefore brings them into optimum relationship with men and events only at those times. In these " states of intellectualized emotion dominated by a fixed idea " (Ribot) they are enabled to alleviate their desire for fulfilment, their purpose is brought into focus, and ability and energy are released which in other circumstances are checked at their source. They are, in fact, reacting to " key experiences ". Types such as these are not readily adjustable to ordinary conditions, and it has been said of them that " such men, though doomed to destruction by the timid herd, grow after death to the full proportion of their greatness ".

In a case of this kind the causes of discord need no description.

A more emphatic, though less permanent, type of reaction is seen in lads who embark on a course of active resistance to discipline which they are well aware is to their disadvantage and contrary to reason. This courting of punishment or disfavour may, of course, have rational but ulterior motives in other cases, such as attempting to obtain a change of location or labour, for example, but in the condition alluded to here there is a vicious circle sometimes imposed on a paranoid psychopathic background for which a means has to be found by which they can extricate themselves. Vigorous methods of dealing with this situation are apt to lead to overt antagonism and inconsistency in conduct in the extravert, which may prejudice his training, or, in the introvert, produce a degree of resentment which may have permanent anti-social consequences. Adolescence is the period of life when the urge to give expression to individual standards of conduct, unmodified by convention, is at its strongest, the result being, in many cases, a failure to effect a compromise between the utilitarian and the ideal points of view.

An example of the vicious circle was seen in the case of a lad (C. L—, æt. 17) who had served about twelve months of his sentence at a Borstal institution before he came under observation. On reception there he had given a good impression, but his attitude underwent a rapid change, and from being frank, willing and amenable he became violent, hostile, defiant and threatening. He resented criticism, was insolent and insubordinate to those in authority, he refused work and courted punishment. In the intervals between his outbreaks he admitted that he was wrong, but this had no effect on his subsequent conduct. His behaviour became so unreasonable that it was obvious that nothing further could be done with him in the existing surroundings.

On examination he gave the following history :

He was the elder of two legitimate children. An older illegitimate son was the favourite in the family, and the lad resented the way that his parents upheld this son. He grew stubborn and difficult, and came to be regarded as the black sheep of the family. This led to his being blamed for offences which he had not committed. On one occasion he was punished for stealing a shilling which he asserted that his sister took, and he remained resentful of the punishment he received.

At the age of 10 he succeeded in obtaining a scholarship, but at the secondary school he got the impression that he was looked down upon as a working-class lad, and he started truanting. For this reason he was sent to an industrial

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school, without, in his opinion, any attempt being made to find out the cause for his misconduct. There he was frequently thrashed for bed-wetting—a long-standing complaint, from which he still suffers. He accordingly absconded and went home.

At the age of 14 he had a severe head injury. He became more wilful and reckless, associated with a gang, and was ultimately convicted and sentenced to Borstal detention. This sentence he regarded as unjust, as he was never tried on probation.

On his reception at the Borstal institution he disliked the nature of the work he was given, feeling that he merited a more trustworthy job. He resented being teased by other lads, though his arrogant attitude and quick temper provoked it. He believed that he was picked out by the leaders for unfair treatment, and he objected to those in his own position being put in authority over him. He thought that his friend did not treat him with sufficient respect in public.

His conduct led to his becoming avoided by other lads, though they were not averse to suggesting fresh forms of defiance for him to carry out, and he was unwilling to risk their taunts. He sacrificed a trustworthy job as he felt unable to withdraw from the attitude he had adopted.

At first he derived some satisfaction from the notoriety which a rebel is apt to acquire, but by the time he realized that he was merely a butt for others and was, in fact, performing for their amusement, he had gone too far to return without sacrificing more of his self-esteem than he was prepared to do.

His desire for recognition was, he felt, being thwarted by his having to be one of a crowd, by his subordination to senior lads and by the way he was treated by his friend.

The position was aggravated by the fact that he had a talent for entertaining, which gave him opportunities for asserting himself. The forces impelling him were, however, too strong for him to keep his self-assertiveness within reasonable limits, and this naturally provoked the opposition of other lads. To justify himself he responded by open defiance of authority, for which he was punished. He found that punishment relieved him of the necessity of supporting his reputation in the former way, and it also added fresh fuel to his feeling of injustice. The probability of his escape from his position by continued misconduct next occurred to him and a transfer to another institution became his objective. This was attained, and after a few weeks of semi-isolation there he abandoned his previous attitude and ultimately returned to complete his Borstal training.

He served the full sentence, but his conduct, did not improve. Shortly after he returned home he enlisted in the Army, but a few weeks later he overstayed his leave, stole money from a gas-meter, and his Borstal licence was revoked.

After a further period of special training he again went home, where he was soon convicted of housebreaking. A year later he was sentenced for stealing.

He then rejoined the Army, making a false attestation, and was doing extremely well until his antecedent history came to light when he was discharged.

Nothing further has been heard of him.

Normal periodicity may be seriously affected when it is disturbed by a mechanism described by Mercier as parasitic, in which the piling up of one

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imperative idea on another of the same kind occurs, until a state of supersaturation exists with its attendant discomfort. Relief is only obtained by an act which may be contrary to the intelligence and volition.

These compulsive ideas, which are manifested in numerous ways, recur at intervals which may be related not only to the rhythmic changes in the body and mind, but also to such periodical conditions as the climate and season. Obsessions have been classified into three groups—those of indecision, fear, and irresistible impulse. All these processes show, in one way or another, comparable periodic features. Thus the rituals which are not infrequently seen in the latter type, following more or less closely a regular pattern essential to the full development of the process, are other instances of the control which rhythm may attain when imposed on abnormally susceptible persons.

The following is a case in point :

XYZ, charged with an indecent assault on a female and common assault. The family history was negative.

This man obtained a scholarship from an elementary school and received a secondary school education up to the age of 16. He then became a Government employee, and having passed the necessary examination for promotion was earning £5 a week at the time of arrest.

He sustained a cut head in a motor accident some time before, but there were no adverse sequelæ, and he had had no serious illnesses. There was no history of alcoholism, epilepsy or insanity, and he denied having had venereal disease.

He gave a history of masturbation beginning at the age of 20, after consorting with a girl of 13, and the habit had increased in frequency during the two years before arrest. Shortly after this event he committed an assault on a girl by whipping, to which she consented, and the charge was brought when this sadistic practice had been repeated a large number of times with five other girls. Coitus did not take place on these occasions. The whipping or its imaginary substitute became a necessary part of the masturbatory act.

In the course of time he had evolved a ritual consisting of an exact formula in words and actions, which he caused the girl to follow at each whipping, though gratification of an almost equivalent degree was obtained by having the verbal formula repeated to him over the telephone.

The sentence used was a long one, and both actors had to be word-perfect if the desired result was to be obtained.

The dependence on a particular sequence of words is indicative of the necessity for some additional recurring factor to complete the process once it has been initiated. This factor may be an extraneous one. The phrases used here are not only for the purpose of recalling a pleasurable memory, for the fantasy itself could be revived by any similar series of words. As in the present case, however, these alone appear to be incapable of ensuring consummation.

Fully developed compulsive states are not very frequently found among adolescents. In spite of the fact that they are of congenital origin in certain

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cases and the propensity is observed in childhood, they rarely make their appearance until maturity is reached. Claustrophobia, although a condition likely to be precipitated by cellular confinement in susceptible cases, is rarely seen at this age in prison, and even abreactions such as those resulting in smashing up cell furniture, which might in some cases be associated with claustrophobia, are rare.

Among the reaction types to which a final allusion should be made are the schizoids and cycloids. The schizoids represent the extreme introverts, whose reaction at their failure to bring their environment into conformity with their needs, instead of adapting themselves to their environment, may be so severe as to compel them to seek satisfaction in a life of fantasy, terminating possibly in a fully developed schizophrenia and such rhythmic manifestations as stereotypy and verbigeration. The cycloids represent the extreme extraverts, whose periodical fluctuations of mood may become so excessive as to culminate in a fully formed manic-depressive psychosis.

The use of the word " parasite " in compulsive states denoting a consuming of the products of the mind by an invader, if the metaphor be taken literally, offers further support to the view that influences of many kinds which are in fact extraneous not only make their presence felt by the nature of their content but also by their rhythm, for the word " parasite " presupposes appetite, and appetite implies periodicity. The failure of the defence at certain times only indicates the presence of alternating strong and weak elements in it.

Enough has been said to indicate the extent and variety of conditions liable to a periodic rise and fall, and it would appear that voluntary effort may to some extent adjust the temperamental habit of the individual in this respect, so that conduct is not incompatible with the environmental demand. Failure to bring this about is one of the many causes of situational maladjustment.

The application of the foregoing thesis to the problems presented by the institutional treatment of adolescent offenders requires a brief allusion to certain points in the conduct of the institutions themselves.

It may be said that in situations where there is continuous control and close organization in every detail, the tendency to lessen the differentiation between one individual and another can hardly be avoided. While the trend of modern penal methods is to approximate conditions in prisons and institutions more closely to those of the social system at large, it is nevertheless true that where the segregation of a number of persons in an artificial environment has to be maintained, there are fewer opportunities for deviation from a prescribed line of conduct. This results in a routine the momentum of which is quickly apprehended, both by the staffs and their charges, by its uninterrupted rotation.

Prison routine, however, offers a solution to the difficulties in not a few types of offenders. The stable, subnormal delinquent is one. Among others

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are the constitutionally inefficient, who are described by East as devoid of initiative but " shrewd, able and willing workers under supervision, capable of appreciating their social obligations, but incompetent to deal with situations which demand the suppression of temporary desires owing to their passive inadequacy to negotiate life according to social conventions and the law's demands ".

This incapacity to carry the responsibility for moral and other decisions is also seen in the reactions of certain drug addicts, rarely found among adolescents, who not infrequently attribute their rapid recovery of poise and confidence, after the immediate symptoms of deprivation have subsided, to the fact that they are not labouring under any obligation to decide whether they will or will not continue to submit to the withdrawal of the drug, whereas in public hospitals and nursing homes they are, in fact, the final arbiters in this matter. This transference of reliance on the support of one external agency to another provides a temporary solution to the conflict between hunger and fear, and results in a rapidly developing but transient form of institutionalization. The agency in this case may be the feeling of security against the consequences of a further infringement of the law, the anonymity of prison dress and the protection it affords, the steady and certain drive produced by the rhythm of the system itself, or more than one of these. Whether the precipitating cause of the addiction be physical or mental or due to contagion, the common attitude in all is a refusal to face reality. That imprisonment is recognized by some drug addicts to provide certain advantages, which public institutions do not possess, in relieving them of this obligation, is shown by letters received from unconvicted addicts requesting advice on the means of obtaining treatment under corresponding conditions.

There is, however, a more deliberate form of institutionalization, such as is seen in recidivists of the Borstal revokee type, who occasionally admit that they prefer the less varying conditions of a special institution to those which obtain at Borstal, because they require less effort to comply with them.

A distinction may be drawn, perhaps, between temporary and permanent institutionalization, for certain recidivists appear to prefer conditions of an unvarying kind while in prison. This reaction is not, however, comparable with that of some factory workers, who are stated to indulge in fantasy as a means of escape from the monotony of certain industrial processes, nor may it be directly compared with those who invariably seek monotonous work in order that their opportunities for fantasy-weaving may not be disturbed, for in other circumstances convicts may not resort to this practice. They differ also from persons of a simple schizophrenic type whose apathetic attitude to circumstances in general results in an absence of effort to resist conditions in any situation. It is, in fact, a form of response attributable to the periodicity of prison conditions.

The search for opportunities to indulge in fantasy-making may be assisted

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by auto-erotic acts, as the following case of masturbation in prison, for the purpose of recalling a fantasy, suggests:

E. G. K—, æt. 20.

This lad's father died in a mental hospital, where he was undergoing treatment for an alcoholic psychosis. He had previously deserted his wife.

The lad was of slightly inferior intelligence, with a mental age of 13 years 4 months, but he was not considered to be certifiable under either the Mental Deficiency or Lunacy Acts.

He admitted that his offences had all been of a sexual nature, though his previous charges had generally been of another kind, and he was inclined to talk extravagantly and boastfully about his sexual exploits to compensate for his belief that he was regarded as an outcast.

He stated that he had an irresistible attraction for sexual excitation, and he had indulged this by auto-erotic, homosexual and heterosexual means.

He had been discharged from the Army for sodomy, which he admitted, together with other similar acts, his explanation for his conduct being that he either did not like to refuse requests, or he needed money.

He cohabited with several woman at various times, and he states that he practised masturbation in prison primarily in order to excite heterosexual fantasies, so that his attention might be diverted from other less attractive matters.

An instance of another type which has difficulty in adjusting itself to conditions of life in the general community is that of the formalist, who derives satisfaction from perfecting routine collective movements, and is, therefore, in accord with the precise daily round of a training establishment. He is alert, bright and conscious of his efficiency in such surroundings, and makes an excellent and co-operative inmate because it would appear that he is relieved of the responsibility of varying his response and of calculating his movements in advance. Although such cases may pass through their course of training with credit, the advantage of applying expert psychological direction to some of them is recognized as a protection against their subsequent failure to adjust themselves to the changing situations of ordinary life, for the mechanisms involved in this type of reaction are archaic in form and are distinct from the higher functions of volition. This tendency, termed "hypobulic" by Kretschmer, may be found in training institutions where mechanical forms of discipline have not been reduced to a minimum. The fact that this can be brought to a state bordering on automatism may be observed in certain of the continental training schools of young offenders.

The general attitude to the community and the demeanour of lads who have had many years' institutional life during the pre-adolescent period are often distinguishable from those coming from ordinary home surroundings, but though it is rarely as recognizable among ex-Borstal inmates who have had no previous experience of institutional life, certain features common to both are observed in their general reactions.

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This effect of corporate training over a period of years is probably due to the stimulation of conscious co-operation towards the welfare of the community as opposed to the less canalized development of instinctual tendencies as a whole which takes place in varying degrees under ordinary conditions, and includes those dispositions which are directly or indirectly concerned in building up the fabric of social adaptation.

Conscious co-operation, it is thought, is developed in proportion to the imitative capacity or suggestibility of the individual, and while this tendency in conjunction with the gregarious and sexual instincts is necessary to normal social development, as Gordon has pointed out, it is not alone able to actuate a system of ideas capable of being applied to all kinds of situations with adequate success. A crowd will resort to actions which the several persons composing it would not contemplate were it not for the fact that the imitative tendency of each individual is stimulated to an abnormal extent by the proximity of others.

The training in social habits of mental defectives has necessarily to rely on imitativeness to a much greater extent than is the case in normal persons in whom the intellectual approach is available, for the tendency to imitation and mimicry is often a well-marked feature, even in such low-grade types as the microcephalics and mongols, both of whom may show an amenability to this form of training which outstrips their mental capacity. The social adjustment of higher grade defectives is often successfully brought about by this means, and is adequate so long as they do not come in contact with situations for which they have no direct precedent. Imitation implies repetition, and this feature is frequently present in the offences committed by defectives. It is of interest also to note that a sense of rhythm even at the auditory level is well developed in certain types of the same class.

The institutional training of adolescents coming within the range of normality will result in a levelling-down of conditions to the point which is attainable by the lower or more retarded grades within that range unless effective classification is ensured. A certain proportion of these inferior types has undergone an institutional upbringing from an earlier age, and owing to the poverty of their experience of the varied situations of ordinary life, are not infrequently mentally retarded. This limitation of mental equipment shows itself more particularly when they are called upon to meet situations to which they are unaccustomed. The following is an instance of this:

F. P—, æt. 17. Convicted of stealing. Columbian group test 50%.

He was in fair physical health, although he was undersized. His mental age was about 14 years, and he showed no sign of insanity, mental deficiency or psychoneurosis. He was brought up in an orphanage, in which he was deprived of playing organized or unorganized games, and suffered severely from his unsympathetic surroundings, which resulted in a sad and repressed manner and facial expression. In common with many other inmates he made repeated attempts to run away.

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When he left the orphanage he was totally unequipped for life in the general community. He had no friends, and when, after losing his hotel employment, he obtained lodgings with a woman who was a receiver, he soon embarked, with her encouragement, on the present series of thefts. He realized he was badly entangled, and expressed his relief at arrest and sentence to Borstal detention. He was anxious to do well in training and to join one of the Services on release.

The lack of sentimental attachments and affective contacts in general and the absence of opportunities to develop his mental mechanisms in the normal way were regarded as strong predisposing factors.

It should be noted that adventurousness, which is a not infrequent factor in adolescent crime, forming about 10·2% of all acquisitive offences, and is a normal response to instinctive urges, tends, through the lack of co-ordinating experience, to be expressed in ill-advised and incongruous ways if the normal outlets for these urges have been withheld in childhood.

Another aspect of institutional life tending to foster imitation is attributable to the fact that the ethical code of those who carry out the training of inmates differs in many respects from that of their charges. In the poorer districts, for instance, stealing from the home is regarded as a serious offence, while the appropriation of the property of other people, especially if it is apparently unwanted, may be connived at. The view that all stealing is unethical is regarded as a peculiarity of certain classes and outside everyday practice in their own. Sedulous compliance with this standard of conduct is, in certain cases, considered, in good faith, to be a temporary necessity, to be observed only while they are under institutional care, and its abandonment on their return to the normal mode of living is accomplished without any feeling of fraudulence.

But, as has been pointed out, there is a third factor to be taken into account in the development of social habits, that is to say, the normal evolution of the sex instinct, which adds that degree of extraversion without which social adaptation is incomplete.

It is significant that abnormal sex practices, such as masturbation, especially when carried to excess, foster introversion at the expense of extraversion, and thus tend to interfere with the capacity for social adjustment.

An instance of this is to be found in the following case :

B. Q—, æt. 18 years 9 months. Convicted of sodomy and failing to observe the conditions of his licence from an approved school, and sentenced to three years' Borstal detention.

The physical condition of this lad was satisfactory, and his intelligence was normal, the Columbian group test being 71%. There was no sign of insanity.

His parents were in domestic service and no home had been maintained.

He was of a roving, restless, introspective and solitary disposition, and was much concerned about masturbation, which began at the age of eight. For many years this had caused him anxiety, limited his aspirations and hindered his relations with other people. His attempts to overcome it had failed, and

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as a result of this and of his feeling of inferiority he had abandoned a strong desire to become a priest, although he still reacted quickly to a religious atmosphere.

His literary and musical tastes were good, although his attainments in other ways were mediocre.

He was frank, truthful and fairly reliable in his statements, and he was clearly in need of advice and help to develop his character and encourage a more social and extraverted attitude to life. In this he was anxious to co-operate.

Although corroborative statistics cannot be produced, there is no reason to suppose that masturbation is more common in institutions for adolescents than elsewhere, although the epidemics of it which occur are apt to give an exaggerated impression of its frequency, and it would appear that so long as the stimulus provided by a training community is vigorous, the tendency to fall into vicious habits is lessened. These habits, however, like other less unwholesome traits, are more contagious in institutions owing to the abnormal stimulation of the tendency to imitation obtaining there.

The fact that the majority of inmates, in spite of their segregation, are better fitted by training to take their place as normal social units in the general community would appear to be due partly to the fact that the development of the sex instinct can proceed normally for a time in the absence of the usual heterosexual contacts, and partly, among other grounds, to the presence of sound parental attachments. Cases are, however, met with in this period of life in whom instinctive impulses, of which the dominant one is generally sexual, with their accompanying emotions, exert an unusually vigorous pressure on the personality, and where this is so, or the impulses are insufficient as in the melancholic, disproportionate as in the hysterical, or inco-ordinate as in the schizophrenic type, the social stimulus of the institution may be unable to redirect them.

Statistics show that the socializing of those whose endowments are not grossly abnormal is, in fact, brought about under appropriate institutional conditions when these are assisted by discerning individual attention and maintained for a sufficient length of time. Thus, just as the extravert can be encouraged to practise deliberation and so cultivate the inner life of the mind, and the introvert to adapt his mental life to the practical demands of his environment, so may discriminating and sympathetic oversight ensure stability of environment or kindle affective contacts for those whose lack of one or the other has led to antisocial conduct during the critical period of adolescence.

The paths by which the various mechanisms of the mind are linked together are sufficiently wide to admit stimuli which can rarely be identical both in substance and in the circumstances in which they arise, but when once the impulse has begun its course along a particular route, the end-result is generally the same. A stimulus, however, though moving in one direction, is not, as it were, homomalous in form, but is faceted in various ways, each individual

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surface having a slightly different implication from the others. The function of temperament appears primarily to be the selection of one or more of these, and it is in connection with this that the lattice-pattern already alluded to can be applied as a working hypothesis, for it is suggested that the design and its angle of inclination determine the aspect which is to be admitted into the mind.

It is thus possible to offer an explanation for some of the inconsistencies observed in the conduct of adolescents suffering from temporary physiological imbalance.

Temperament, however, is not a quality which lends itself to detailed classification, and Shand's interpretation of it as "that part of the innate constitution of the mind which is different in different men, so far as this refers to their feelings and perhaps also to their wills", permits the necessary latitude for this statement. The gauging of temperament, nevertheless, is a valuable weapon in the armamentarium of the investigator of a youth's reactions in the past, and may indicate the means to be adopted to bring about the rehabilitation of some of those who, for obscure reasons, have become delinquent.

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ENDEMIC BACILLARY DYSENTERY.

By J. J. B. MARTIN, M.D.

SPORADIC bacillary dysentery is an accepted concomitant of lunacy, and mental hospitals, on statistical evidence, harbour more than 99% of the disease.* When an epidemic of dysentery occurs, the laboratory of the mental hospital is cluttered with fæcal specimens, but when the epidemic is resolved on a solitary "carrier" and a faulty drain the disease ceases to arouse further interest. Little effort is made to assess and control the endemic level. This the author has attempted to do, in a county mental hospital of, roughly, 1,000 beds.

At the outset I wish to acknowledge the great help given in the preparation of this paper by the Assistant Pathologist and Laboratory Technician, Mr. O. T. Moore, whose skill and ungrudging labour alone made its completion possible. My thanks are also due to Dr. P. W. Bedford, who instigated and supported the investigation and permitted the publication of this paper.

The following is a summary of the work done over a period of four years :

Fæces : bacteriological examination	2395
Sera : Widal	921
Water : bacteriological examination	221
Gall-bladder, post-mortem bacteriological examination	90
Intestine, post-mortem bacteriological examination	12
Typing, absorption tests	48
Blood : culture	22
Urine : culture	21
Blood : bactericidal test	22
Gastric juice : chemical examination	60
Dysentery vaccine prepared (approx.)	30 litres.

A. ASSESSMENT.

1. *The Extent of the Disease.*

Dysentery being regarded as a sanitary index, and therefore as a reflection of hospital administration, hospital records minimize the extent of the disease. The first "obituary table" of the hospital, published a hundred years ago, reveals that 10% of the total population died from dysentery in one year, and

* *Incidence of dysentery in mental hospitals.*—Dawson (4) in 1921 stated that on statistics dysentery was only 100 times more common in mental hospitals than outside. When making his calculation he failed to deduct the registrations of dysentery given by mental hospitals from the total registrations given by the whole country, including mental hospitals. Actually dysentery is 400 times more common in mental hospitals than outside.

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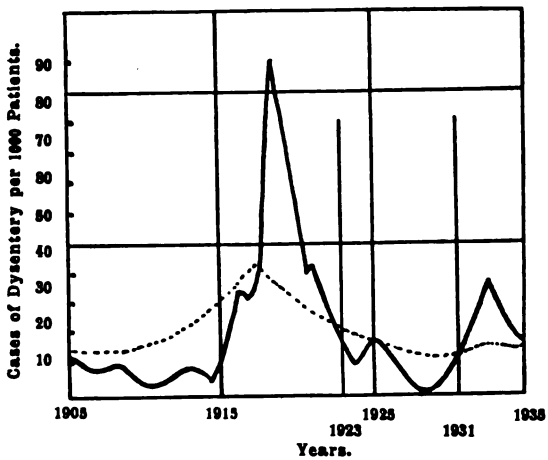
it is a fairly safe deduction that dysentery has persisted in the hospital ever since.

From 1905 onwards all cases of dysentery are purported to be tabulated in an official "register of dysentery and diarrhœa". From this the apparent incidence of the disease is plotted per 1,000 of population (Chart I). The dotted line represents the apparent incidence for all mental hospitals in England and Wales.

This graphical picture of the disease is far from accurate. From 1905 to 1923 the figures depend on arbitrary clinical diagnosis, and only epidemic groupings are shown. From 1923 (when a laboratory was established) until

CHART I.—*Incidence of Dysentery.*



1931 the figures represent an opposite extreme: only those cases which were obvious clinically and which proved to be bacteriological positives are included. From 1931 to 1935 the figures represent an intensive effort to expose dysentery in every manifestation of the disease. Of the last 80 cases recorded, 29 (or almost 40%) were sub-clinical or symptomless, and the proportion of symptomless cases continues to rise. As an old nurse said, rather irritably: "There's far less dysentery, but far more fuss."

Serological incidence.—Even the figures of recent years represent only a fraction of the real extent of the disease. Below, two very graphic serological pictures are presented, side by side. On the left (Table A) are the agglutination figures of an average ward of 47 patients. Two cases of dysentery occurred in this ward; two further cases ("carriers") were found, and a routine bacteriological and serological examination was made of all cases. The examination extended over two months. On the right (Table B) is a

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TABLE A.—47 *Female Chronic Patients.*

Agglutinins.					Fæces.
V.	W.	X.	Y.	Z.	
100	25	25	25	50	—
25	0	13	12	25	—
50	20	12	10	25	—
30	12	13	10	12	—
50	20	25	10	25	—
40	0	0	5	0	—
20	10	12	10	20	—
25	0	0	12	10	—
10	0	0	10	0	—
12	0	0	12	0	—
(25	0	0	8	12	±)
50	10	20	20	20	—
12	0	0	0	0	—
12	0	12	8	0	—
5	20	10	10	8	—
25	0	40	16	40	—
5	0	0	5	0	—
15	35	25	25	12	—
20	10	10	8	0	—
12	12	12	30	0	—
50	0	20	40	40	—
15	0	0	5	0	—
35	0	20	30	25	—
25	0	12	8	25	—
12	0	0	4	12	—
25	0	0	8	25	—
12	0	0	4	25	—
25	0	12	30	0	—
100	25	25	21	25	—
5	0	0	0	0	—
25	0	12	8	12	—
5	0	0	6	12	—
12	12	0	8	0	—
35	0	12	13	20	—
25	0	0	0	0	—
35	0	0	8	12	—
35	12	20	21	25	—
25	12	12	8	12	—
50	0	40	21	62	—
(25	0	20	21	12	+)
25	0	20	8	12	—
20	0	0	8	0	—
12	0	40	42	62	—
25	12	0	13	12	—
12	25	12	21	12	—
10	18	0	0	12	—
50	12	62	25	62	—
25	6	12	13	16	

TABLE B.—47 *Female New Admissions.*

Agglutinins.					Fæces.
0	0	0	0	4	—
0	8	0	0	4	—
0	8	0	10	4	—
0	0	0	0	0	—
0	0	0	0	0	—
16	0	0	0	0	—
0	0	0	4	0	—
0	0	0	0	0	—
0	0	0	0	0	—
0	0	0	0	0	—
0	0	0	0	0	—
25	0	0	0	0	—
0	0	0	0	0	—
0	8	0	0	0	—
0	0	0	0	0	—
0	0	0	4	0	—
0	16	20	8	25	—
6	0	0	0	0	—
12	0	0	0	0	—
5	0	0	0	0	—
5	0	0	0	0	—
5	0	0	0	0	—
12	0	0	0	0	—
5	0	0	0	0	—
12	12	16	12	12	—
12	25	16	0	12	—
5	0	0	0	0	—
12	0	0	0	0	—
20	0	12	16	12	—
6	0	10	8	12	—
12	12	0	8	12	—
3	0	0	0	0	—
50	12	25	8	12	—
12	0	0	8	0	—
50	25	12	8	0	—
5	0	0	8	0	—
12	0	0	4	0	—
12	0	0	4	0	—
2	0	0	4	0	—
5	0	0	0	0	—
5	0	0	0	0	—
25	12	0	0	0	—
12	12	12	8	12	—
12	0	0	4	0	—
12	0	0	8	0	—
12	0	0	6	0	—
35	0	0	8	12	—
9	3	3	3	3	

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comparative series of consecutive admissions to the hospital, taken about the same time. The agglutinations were performed against the standard Oxford suspensions. The readings were taken at four hours. The reduced titres are shown.

The contrast between the chronic patients, long resident in the hospital, and the new admissions is clear. The new admission, in the vast majority, is a serological negative. The chronic patient shows the full serological spectrum ; and in the average hospital ward, if active infection is momentarily confined to a small proportion of patients, over a period of years the Flexner antigen enters into 100%.

Under present hospital conditions the insane population makes an ideal breeding-ground for the disease. Dysentery is rarely imported into the hospital. Only one " carrier " has been found in the routine examination of over 500 new admissions. The source of infection is within the hospital walls, and the endemic level is never zero.

2. *The Causal Organism.*

In many endemic areas, and in some mental hospitals, the types of infecting organisms are extraordinarily numerous. In this hospital one particular species, *B. dysent.* Flexner, is responsible for 96% of dysentery, and the organism recovered is invariably of one sub-type, which may be designated Flexner Zx.

The cultural characters are largely as described in standard works. The colony has few distinguishing features. Growth is unaffected by mild changes in the H.I. concentration of the medium. It grows freely in bile, in whole blood or citrated blood, but less freely in serum or dilutions of serum. Rough colonies have not been obtained from original platings. The sugar reactions are constant : dulcite is never fermented. No organism giving the typical cultural reactions was found to be inagglutinable by specific sera or vice versa.

The toxicity of the germ appears to be constant. Typical dysenteric stools follow an excessive dose of the killed suspension, and in one subject who died of intercurrent disease a day after inoculation the intestine presented the appearance typical of the disease. On the other hand, the virulence of the organism, as measured by its power to infect, to cause disease, and to kill, shows a wide variation. Clinical variations in infectivity cannot be altogether explained in terms of the host and his environment, or in terms of dosage. A clean patient, in a ward with good hygienic conditions, excreting the germ only occasionally, and in small numbers, may precipitate a series of cases. A dirty patient, in bad hygienic conditions, may excrete large numbers of germs persistently, without infecting others. Similarly, while it is plain that rapidity of passage increases, and a high degree of specific immunity diminishes the severity of the individual case, this relationship is not constant.

Other organisms.—Morgan's No. 1 (28, 29, 30, 31, 32).—This organism was first described by Morgan in 1906. The cultural and biochemical characters are :

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Glucose A → G, lactose O, mannite O, dulcite O, sacharose O, milk O or A → Alk., indole +, motility + or O.

We have found this organism in normal fæces and in solitary cases of diarrhœa in old and debilitated subjects. Although Shaw Bolton (1930) regards this organism as specific, or as so intimately associated with true dysentery as to be potentially specific, we have not found it related to any serious case of diarrhœa, or to any series of cases, nor have we found any association between it and true dysentery.

Douglas and Colebrook No. 8 (33, 34, 35, 36, 37).—This organism belongs to the *B. alkalescens* group. It was first described by Douglas and Colebrook in 1917, and labelled *B. dysenteriæ* Andrewes in 1918. The cultural and biochemical characters are :

Glucose A, lactose O, mannite A, dulcite A, saccharose O, milk Ac.-Alk. +, indole + ?, motility O.

It is distinguished from Flexner in the following respects :

(i) It is a late fermenter of dulcite. This feature is invariable, although fermentation may be very late (10 to 14 days).

(ii) Although perhaps at first faintly acid in milk, the ultimate reaction is strongly alkaline.

(iii) The production of indole is inconstant, whereas the production of indole by *B. Flexner* is always demonstrable by a suitable technique.

(iv) It is inagglutinable by Flexner sera and vice-versa.

We have found this organism :

(i) In the normal fæces of healthy patients.

(ii) Alternating with *B. Flexner* in subacute cases of dysentery ; fæcal specimens and sigmoidoscope swabs.

(iii) It was found more commonly in a ward where dysentery (Flexner) was active ; 14 out of 70 consecutive specimens.

(iv) In solitary cases of diarrhœa, where no (other) causal organism was found, particularly in old and debilitated patients.

We therefore had some reason to suspect the organism as pathogenic and its frequent association or alternation with Flexner at one time made us wonder whether it was not an inagglutinable variant of *B. Flexner*. The pathogenicity of the organisms seems to be very doubtful. It is rarely found in pure culture, like dysentery, from a loose stool. Its toxicity is slight ; the symptoms (a dangerous criterion, maybe) are never severe or lasting. The organism disappears rapidly from repeated specimens. It is not agglutinated by the patient's serum at any time. And finally, although patients excreting the organism have not been isolated or subjected to any special precautions, we have not known even two definitely related cases, such as we might expect to find if there were any potential epidemicity.

We have attempted to approximate Douglas and Colebrook No. 8 to Flexner in various ways. We attenuated Flexner by heat ; we grew Flexner in immune sera ; we educated it to strongly acid and strongly alkaline media ; we coaxed and cajoled it, but it refused to ferment dulcite. Nor would Douglas and Colebrook respond to the reverse form of education.

Our opinion is that the biochemical characters of Douglas and Colebrook No. 8 cover not one, but a group of allied organisms, often symbiotic with the Flexner group, which can be safely disregarded as primary causes of epidemic diarrhœa.

B. Sonne (38), although theoretically interesting as a comparatively new disease with a healthy exotoxin and a beautiful S—R variation, accounts for

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only 4% of cases since it was first discovered in the hospital in 1929. It seems likely that the disease was introduced from the surrounding districts; Sonne is endemic in the villages near the hospital. Despite the liability to importation, Sonne shows no tendency to spread in the hospital population; and according to serological evidence it has not been responsible for any high proportion of disease in the past history of the hospital. The organism now holds few bacteriological mysteries. The cultural reactions are fairly distinctive; the agglutination of the suspect organism is clean-cut; the sera of suspected patients show rapid and distinct agglutination curves; and a serological positive, in contradistinction to *B. Flexner*, is sufficiently rare to have some value in diagnosis.

Path of invasion.—The path of invasion is a matter of conjecture. On the analogy of other organisms pathogenic to the bowel, such as *B. typhosus* or *B. ærtrycke*, the path of invasion would be indirect: absorption through the more permeable mucosa of the upper alimentary tract, bacteriæmia, excretion of the organism in the bile, and secondary infection of the lower bowel. In clinical fact a bacteriæmia is rarely demonstrable, and the organism has never been recovered from the bile. The usual assumption is, therefore, that the path of invasion is direct, by simple ingestion.

Distribution of lesions.—Whatever the path of invasion, the initial lesions of the acute case are not localized, but are distributed throughout the length of the colon and the lower third of the ileum. The chronic lesions are equally diffuse, but pus-pits and mucus cysts are more probable in the lower colon. The mesenteric glands draining the infected area may harbour the organism, but there is no focal residue in the gall-bladder. Urinary excretors are rare.

3. *Symptomatology.*

According to degree of symptoms, there are four distinct types of cases: symptomless, sub-clinical, subacute, and acute.

(i) *Symptomless.*—The organism may be excreted without signs or symptoms of the disease. The organism is ingested, passes through the body, and may be recovered from the fæces. The host is unaware of its presence, and the stool is normal, both to macroscopic and microscopic examination, with a complete absence of cellular mucus.

(ii) *Sub-clinical.*—

(*a*) The stool contains shreds of muco-pus.

(*b*) One loose stool, containing muco-pus, and, perhaps, blood.

(*c*) Mild, transient diarrhœa.

(iii) *Subacute.*—There is diarrhœa, lasting a few days, with slight malaise and slight pyrexia.

(iv) *Acute.*—This is the typical picture of the disease: a short incubation period, with malaise; a rapid pyrexia, sometimes preceded by rigor; usually vomiting; then diarrhœa, accompanied by griping pain and followed by

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tenesmus. The stool contains blood, mucus, epithelial debris, inflammatory cells and organisms.

Chronicity.—There is a wide variation of chronicity, and the severity of symptoms is a bad gauge of the duration of the disease. An acute phase may last hours, or days, or weeks; a subacute ulcerative process drags on for months, and a retention cyst takes years to burrow to the surface. Ilitch quotes a case observed over a period of seven years, during which the organism was excreted only on 15 days out of 2557, i.e., one positive specimen to 170 negatives. This is, perhaps, a somewhat extreme example, but it emphasizes two vitally important points: first, that the excretion of the germ is essentially intermittent, and second, that this intermittent excretion may persist indefinitely.

The carrier state.—There are two distinct types of carrier.

The first is the *transient excretor*: the subject who harbours and excretes the organism for a very short period of time, without displaying any sign or symptom of disease. Of repeated consecutive fæcal specimens, only one is positive. The stool is formed. Blood and mucus are conspicuously absent. Sigmoidoscopy reveals a healthy gut. Such a type of carrier may be found among any infected group. He is an interesting phenomenon, suggesting the presence of many others of his kind, unsuspected and undiagnosed, in whom there is an exact balance between resistance and virulence. He will exist in large numbers during an epidemic, and he becomes commoner if the standard of immunity is raised by natural or artificial means and no measures are taken to minimize conveyance.

The second is the *chronic intermittent excretor*, who has a diseased gut, and excretes the germ intermittently, with minimal symptoms, over a period of years. There is no history of disease, and very often the only visible sign is the presence of cellular mucus in the stool. In the academic sense he is not a true carrier: he is a missed case. It is in him that endemic disease drags on, for 5, or 10, or 20 years, and no hospital can consider itself free from disease until he and all his kind have been completely eradicated.

4. *Conveyance.*

Viability of organism.—B. dysent. Flexner has a power of survival outside the body which is not generally recognized. An agar slope survives for months. The organism is easily killed by heat, but it will live for long periods in ice. Variations in humidity have little effect; it may live in sterile water for a month, and, although stated to favour a moist medium, it may survive drying for 10 weeks. Although the difficulty of recovering the organism from a stale specimen is notorious, it does not die quickly in fæces. According to personal experiment it survives in an impregnated fæces for 11 days;*

* A dysenteric exudate was impregnated with an autogenous culture. It was therefore much more heavily infected than a normal dysenteric stool, and the 12 days of viability represent a probable maximum time of survival, rather than the average time of survival.

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according to the literature it has been recovered from dejecta on soiled linen after 30 days, from the garden soil after 49 days, and from the polluted earth of a military camp after one year. It is, however, very sensitive to light, being killed by exposure to bright sunlight after half-an-hour.

Habits of population.—In a mental hospital the path of the organism from anus to mouth is made peculiarly easy by the degraded habits of many of the patients. A group of 431 female patients was analysed thus :

	Number.	Percentage.
Definitely faulty in habit	86	20
Sometimes faulty in habit	75	17
Clean	270	63
Total	431	100

This same group of patients is distributed through ten different wards. Only two wards contain 100% of clean patients ; only those two wards have remained free from dysentery in the past 5 years. Of the last 80 cases of dysentery, 46 are of faulty habit.

Inadequate sanitary facilities.—Such a degraded population requires a very highly elaborated system of sanitation ; in the hospital to which these investigations refer this did not exist. The water-closets were inadequate in type and number ; there was a widespread use of chambers and old-fashioned commodes ; poor facilities were available for the disposal of soiled linen. Baths and running water were at a premium. The hospital structure is old-fashioned and not designed for cleanliness.

Overcrowding.—The hospital is often overcrowded. Not in theory. In theory each patient has his allotted quota of day-space (440 cub. ft.) and night-space (550 cub. ft.). This hospital has, theoretically, empty space for 200 extra beds. And yet at one time one might find a ward where, if all the patients sat down simultaneously, they were virtually touching each other, and where the beds, at night, were less than 1 ft. apart. The hospital at present accommodates 900 patients. Postulate an arbitrary maximum at a lower figure (800), and plot the curve of excess population against the dysentery index (Chart II). The similarity of the two graphs is too remarkable for sheer coincidence.

Subjective factors determining invasion.—A mental hospital population has little power of resistance to disease. The standard of the average patient is C3 ; dysentery picks out the C4 fraction. His food is deficient in protein value and in vitamins. The integrity of the mucous membrane of the alimentary tract is vitiated by a variety of causes : bad teeth ; imperfectly masticated food ; deficiency of secretions ; constipation and its corollary, excessive doses of salts and purgatives. 50% of patients show obvious signs of intestinal catarrh ; and the patient with feeble powers of general or focal resistance has

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little chance of acquiring a specific immunity, however prevalent the disease. An attack of the disease does not preclude reinfection. There is a floating

CHART II.

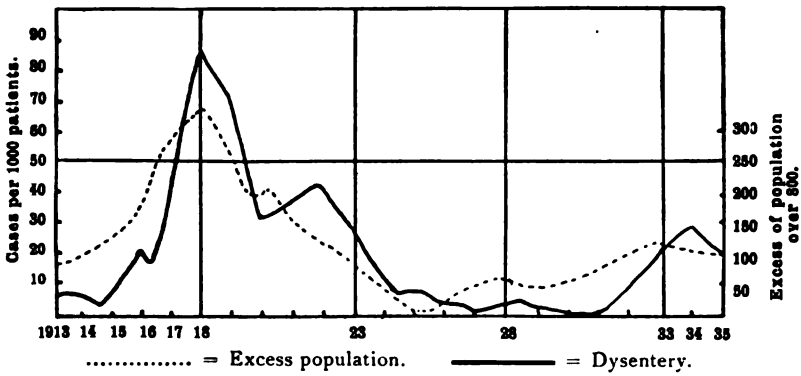
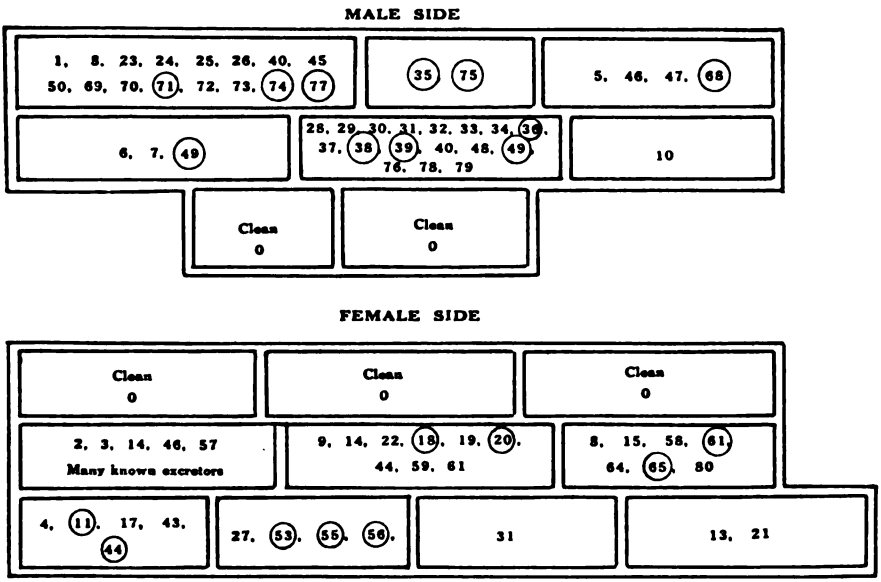


CHART III.



population of all grades of immunity, and a regular influx of new admissions—perfect soil for the disease.

The endemic nucleus.—The number of infectors is very much larger than the number of actual cases of dysentery would suggest. In Chart III is given

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a rough diagram of the hospital, divided into wards, and on this diagram a sequence of cases is plotted ; symptomless excretors are marked with a ring.

These cases occurred consecutively over a time of only four years, following a period when the hospital was, supposedly, free from disease. Yet dysentery is widespread ; more than two-thirds of the wards have been infected, and symptomless excretors have been discovered in 9 of the infected wards.

Spread.—The spread of the disease is at first slow, subterranean, and confined to the infected ward. But the segregation of the mental patient in his ward is far from absolute. Presently an infector is transferred, undiagnosed, to another ward, which in its turn becomes a focus of disease. Or worse, an infector is turned loose, as a worker, in the hospital. Active excretors have been found working in the hospital kitchen, the stores, the farm, and the garden.

Remote dissemination.—If active excretors are distributed all over the hospital, an epidemic is no longer a risk, it is a certainty. Sooner or later the organism will find its way into some common medium—water, or milk, or bread. With the outbreak of a large number of simultaneous cases, control becomes impossible. Diagnosis becomes guess-work. Complete isolation is impracticable. Treatment is imperfect. And when the epidemic subsides, practically the total hospital population is suspect of residual infection.

B. Control.

Effective control is possible only in the pre-epidemic phase of the disease process. Efficiency will depend on the ability of the hospital staff to diagnose the disease, to promote healing, to segregate infectors, to minimize conveyance, and to create in its population a power of resistance to disease.

1. *Diagnosis.*

Cases with symptoms.

There are two principles which should be inculcated ***ad nauseam*** :

(i) All fæces should come to the laboratory for examination if—

(*a*) The patient has diarrhœa, or—

(*b*) The stool contains blood or mucus, or both.

(ii) If a patient excretes blood or mucus or both in conjunction with any degree of pyrexia, the case should be considered as dysentery, and treated as such, until proved otherwise.

Cases without symptoms.

In the search for excretors without obvious clinical signs two alternative methods are available.

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1. Routine examination case by case and ward by ward of the total population. This method is not advocated. Unlike typhoid, bacillary dysentery does not depend, for growth and spread, on an almost static number of "carriers". It is a much more active process, and, further, the symptomless excretor is much more difficult to diagnose.* To cover a hospital population of, say, 1,000 patients in any reasonable time would demand a laboratory of immense capacity, and an expenditure of material and labour totally incommensurate with the number of positive results obtained.

2. The alternative method is to wait for a case to display symptoms, and then examine contacts. This may at first sight appear to be a confession of failure; the attack on the disease seems indirect. The method is, however, extremely practical, and it has produced results in the immediate reduction of the disease which routine and indiscriminate examination *en masse* did not attain. There is a higher number of positive findings, and even if some of these come from transient excretors, there is a very high probability of the chronic infector being discovered and isolated.

Provocative vaccine.—To stimulate the latent excretor, a provocative vaccine may be used. The utility of such a vaccine was suggested in the course of routine inoculation of the hospital. Given an adequate dose, the latent excretor shows active signs of disease, and infected mucus is excreted in the fæces. Our practice is to give 25 millions intravenously.

Macroscopic examination of stools.—In the examination of contacts, it is almost useless to plate any stool not containing mucus. Whitehead and Kirkpatrick plated 5,000 stools not containing mucus to find 0·1% positive, and 100 stools containing mucus to find 75% positive—a ratio in proportionate infectivity of 750 to 1.

The whole stool should be exposed for examination. It should be passed naturally into a clean commode, drained of urine, transferred to a clean chamber, and covered. Thereafter the stool is spread on a flat dish. If mucus is apparent, several loopfuls are transferred to a tube containing glycero-saline and sent to the laboratory. If mucus is not apparent, a portion of the stool is macerated in sterile water, when shreds of mucus, if present, float out. Significant mucus (muco-pus) is recognizable on the surface of a formed stool. It is a yellow white, opaque, and more friable than simple mucus. Long, stringy mucus is usually negative.

* *Relation of "carrier" to acute cases in typhoid.*—We even doubt the jump from extreme chronicity (the carrier) in typhoid to the acute case. This hospital boasts carriers of typhoid and of paratyphoid B, and out of, roughly, a dozen cases of the disease we have only seen two text-book cases. Among the other ten we have seen and demonstrated:

(i) Typhoid fever without abdominal symptoms, a negative blood culture on the fourth day, and only one positive stool, on the tenth day.

(ii) Paratyphoid without abdominal symptoms, a negative blood culture on the third day, and only one positive stool, on the ninth day.

(iii) Paratyphoid of the pure bacteræmic type, blood positive, and stools consistently negative.

(iv) Paratyphoid with negative blood culture on the second day, no abdominal symptoms, a sore throat, a typical rash and temperature, and persistently negative fæces: the agglutination curve soared from 0 to 5400, clinching what seems, on paper, an absurd diagnosis.

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Taking of specimens.—When the stool cannot be kept for macroscopic examination by a trained observer, a specimen outfit is employed by the nurse. This should be as simple as possible ; a metal spoon, preferably long to avoid soiling the nurse's hands, is embedded in a cork fitted into a wide, flat-bottomed tube containing enough glycero-saline to cover the specimen. The nurse is instructed to include mucus, or blood, if present, in the specimen.

Delay in plating.—It is essential that the specimen, whether taken by the bacteriologist or the nurse, should be fresh, particularly if, as usual, contaminated by urine. Little and Bernstein did comparative tests on a series of cases from acute cases, spreading one plate for each specimen within ½ hour of excretion, and another plate from each specimen after 3 hours ; after 3 hours the possibility of a positive finding was reduced by no less than 50%. Specimens should be timed as well as dated, and specimens more than an hour old should be discarded, if not previously transferred to a preserving medium.

Preservation and enrichment.—The preserving medium employed is glycero-saline, 10% glycerin in normal saline. This medium probably inhibits the normal overgrowth of coliform organisms, and allows the more fragile dysentery bacillus to survive.

Microscopic examination of fæces.—A portion of the mucus, or of the exudate of the acute case, is examined under the microscope for the presence of blood or pus. If pus cells are found, a bacterial process has been demonstrated, and further specimens are demanded from the case. Even if pus cells are not found, we proceed to plating ; if the plating is negative, and there are no suspicious symptoms, the examination of the case is temporarily closed.

Plating.—Plating is done on McConkey's medium, using the largest Petri dishes adaptable in quantity to a small incubator. If there is mucus, it is washed twice in sterile water, then emulsified in saline. A loopful of the emulsion should give an even spread of colonies.

Plates are examined with a lens after 24 hours ; on numerous occasions a solitary colony has been positive. All types of non-lactose fermenters are put in glucose. Where rapid identification is necessary, slide agglutination is useful, but it is unwise to give a negative finding because the N.L.F. refuses slide agglutination. The suspected organism is agglutinated against the standard sera, and no organism giving the typical sugar reactions in glucose, lactose and dulcite is considered inagglutinable until it has been grown in broth.

Agglutination.—The agglutination of suspected sera against the known organism has only a limited value in diagnosis.

(i) It is essential to use specific, not mixed suspensions, of uniform sensitivity, and a rigidly standardized technique.

(ii) A single estimation of agglutination titre has little practical value ; the highest titre may be found in the healthy contact, and a completely

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negative titre in the actual case of the disease. In searching a widely infected group for active excretors a single estimation of each subject is completely valueless.

(iii) An agglutination curve, on the other hand, composed of two or more estimations performed under identical conditions is of practical value in diagnosis:

(*a*) A rising titre plus symptoms, even if bacteriologically negative, is tantamount to proof of the existence of the disease.

(*b*) If, in a case of the disease bacteriologically positive, the titre does not rise, there is a strong presumption that the disease has existed in the subject for a considerable length of time.

(*c*) The value of a curve is modified, but not destroyed, by protective immunization. In every suspected case specimens of blood should be taken at weekly intervals for 3 weeks.

Bactericidal tests.—These were performed according to Thojtta's technique. The serum to be tested was put up in dilutions of saline: $\frac{1}{5}$, $\frac{1}{10}$, $\frac{1}{20}$, and $\frac{1}{40}$. To each tube was added a drop of broth and 0·01 of a dilute emulsion of *B. dysent.* Flexner (one loopful to a standard emulsion of 100 c.c.). The tubes were incubated for 3 hours. 0·01 c.c. from each tube was plated on a 15 mm. Petri dish; the dishes were incubated for 24 hours, and the colonies counted.

The control, without serum, was tubed, plated and incubated in the same manner.

The counts were proportionately reduced to make the control figure 100.

Results.

Dysentery Excretors.

	Agglutinins.	Dilutions. $\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{20}$	$\frac{1}{40}$	Control.
1	63 RT	5	5	6	47	100
2	63	5	6	6	87	100
3	8	6	11	57	91	100
4	0	9	11	28	79	100

Normal Subjects.

	Agglutinins.	Dilutions. $\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{20}$	$\frac{1}{40}$	Control.
1	13 RT	4	8	12	88	100
2	13 RT	14	16	57	109	100
3	0 RT	31	59	93	104	100
4	0 RT	58	104	110	94	100

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Comparative Tests in One Subject.

	Dilutions.				
	$\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{20}$	$\frac{1}{40}$	Control.
Serum	5	5	6	47	100
Defibrinated blood	36	48	100	100	100
Citrated blood	88	109	107	115	100
Serum heated at 55° C.	100	100	100	100	100
Serum absorbed with specific organism	100	100	100	100	100

For an improved technique and a discussion of the problems involved see Mackie, Van Rooyen and Finklestein, *Journ. Path. Bact.*, xxxix, 1934, p. 89; *idem*, *Journ. Hygiene*, xxxii, No. 4, p. 494.

Sigmoidoscopy.—In theory, direct examination of the diseased intestine with the sigmoidoscope should be an indispensable aid to diagnosis, particularly the diagnosis of chronic dysentery. According to Biggam the sigmoidoscope reduces the possibility of a missed diagnosis from 30% to 3%.

The signs to be looked for are four :

(i) Shallow ulcers, sharply defined, but without a raised margin. They are small, rarely larger than 1 cm., round or serpiginous, with a granulating base.

(ii) Umbilicated papules.

(iii) White granulations on a red and thickened mucosa.

(iv) Polypi.

H. J. Smyly states that these signs are present (to the sigmoidoscope) in 93% of cases of Flexner dysentery, and the diagnosis can be clinched by direct swabbing of the visible lesions.

In mental hospital practice results are not nearly so conclusive. Sigmoidoscopy takes a good deal of time and labour : the preparation of the mental patient is peculiarly difficult and he invariably requires a general anæsthetic. In the hands of a colleague (Dr. S. H—) a group of known or suspected excretors showed the following results :

Visible signs of disease, swab positive	5
,, ,, ,, ,, negative	19
No ,, ,, ,, ,, positive	1
,, ,, ,, ,, ,, negative	25
Total	50

The absence of visible signs of disease is obviously of greater importance than the presence of visible signs, and the chief uses of sigmoidoscopy seem to be these :

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1. To establish a test of cure in recent cases when symptoms have subsided.

2. To distinguish between transient excretors and chronic cases without gross symptoms.

3. To eliminate the possibility of the disease in cases showing suspicious symptoms but persistently negative bacteriologically.

The complete diagnosis.—The complete demonstration of a case of bacillary dysentery would include five signs :

1. Positive symptoms.

2. The excretion of muco-pus.

3. The recovery of the organism from the fæces or from the sigmoidoscope swab.

4. Visible lesions.

5. Positive agglutination.

No single sign, of itself, proves the existence of the disease ; all signs are not equally demonstrable ; and immediate diagnosis is not a scientific certainty—it is a matter of probabilities, depending on the combined art of the nurse, the doctor, and the laboratory. If the nurse is negligent, the doctor is ignorant, and the pathologist is blind. The recognition of any one sign demands prompt isolation and treatment. The other signs may be elicited at leisure.

Registration of cases.—There is one essential of co-operation ; that an adequate record be kept of cases, of suspected cases, and of suspicious symptoms. The method of registration advised is threefold :

(i) A hospital register of infectious and potentially epidemic disease : of proved cases only.

(ii) A ward (and possibly individual) register of suspicious signs and symptoms.

(iii) An individual laboratory record for each patient.

2. *Treatment.*

Diet.—This, in the acute stages of the disease, should be minimal, with abundance of fluid. Glucose provides sufficient nourishment for the first few days. Milk and albumen water should both be avoided, but watery arrowroot or Benger's food may be added as symptoms subside. The diet of the chronic case should exclude foods with much roughage, but should have a high protein content to avoid excessive fermentation and a good vitamin value.

We gave an artificial preparation of vitamins A and D to the patients of an infected ward over a period of two months without appreciable effect in raising the general standard of health or diminishing infection.

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On the other hand a glass of milk daily caused a general increase in weight and appeared to diminish infection.

Medicine.—The standard treatment is still the exhibition of some inert salt, usually sodium sulphate. The initial dose need not exceed ʒvj in the first day; this dosage should be gradually tailed off during the next week. The efficacy of the treatment is undermined unless drainage is counterbalanced by abundant fluid, given, if necessary, into the veins.

Specific.—If a potent serum specific to the sub-type of Flexner can be obtained it should be given to any patient severely ill. Such sera being expensive and difficult to obtain, in milder cases an identical antigenic effect can be procured by the exhibition of intravenous vaccine. The initial dose of five millions on the third or fourth day of the disease does not accentuate symptoms; on the seventh day the dose may be doubled and continued weekly over a period. In the chronic case a dose of 10 millions is always safe, and the dose may be worked up to 50 millions. No dose, unless designedly provocative, should accentuate symptoms. The ultimate effect of vaccine treatment, in the acute case, is to prevent chronicity.

Lavage.—No specific treatment, however potent, will break down the avascular fibrous barrier surrounding a very chronic lesion. The essential adjuvant is colonic irrigation, which is really a form of internal massage plus drainage. The solution used need not necessarily be irritating or antiseptic. It should be sterile, warm, and slightly hypertonic. Irrigation should be practised daily on all cases showing visible lesions to the sigmoidoscope until those lesions disappear.

Test of cure.—There is no single test of cure. From the communal aspect, no known case of dysentery should be released from isolation until these criteria are satisfied:

1. The absence of symptoms.
2. The absence of muco-pus from the fæces.
3. Negative sigmoidoscopy.
4. Negative bacteriological examination of sigmoidoscope swabs, and of mucus, if present in the fæces, and repeated negative examination of the fæces.

Our actual procedure is this: The selected patient is given a provocative dose of vaccine—20 millions intravenously—and put to bed for observation. The stool is examined daily for 5 days, macroscopically for mucus, microscopically for muco-pus, and bacteriologically. If these tests are negative the patient is sigmoidoscoped. If no lesion is seen, and the swabs are negative, he is considered as a potential cure, and discharged from isolation.

This procedure may seem somewhat lengthy and somewhat complicated; it requires a certain amount of labour—but it represents, we think, an irreducible minimum, even although the proportion of rejects is very high.

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3. *Segregation.*

The ideal is the rigid division of the entire population into three classes, infectors, immunes and susceptibles, with as great a space as possible between the first and the last. In a mental hospital such a division is not feasible.

Infectors.—There must be an isolation hospital containing two wards.

(i) The first ward contains only known excretors.

(ii) The second ward contains suspected excretors of two types : (*a*) convalescent cases, and (*b*) cases displaying suspicious symptoms.

The second ward will be larger than the first. All patients showing signs and symptoms should be transferred immediately to this ward, and treated as infectious until proved otherwise. The practice of using a sideroom in a general ward cannot be too strongly condemned. The general ward has not the staff or equipment for proper disinfection and isolation, and the most lavish display of cresol and Jeyes' will not prevent spread of the disease. There is only one rule ; isolate at once in the dysentery hospital.

Susceptibles.—(i) The bulk of new admissions must be regarded as highly susceptible. They should therefore be kept apart from the older hospital population. The admission hospital, if it exists, forms an admirable unit for such a purpose. If there is no admission hospital, one ward, or a group of wards, should be set apart for admissions.

(ii) Transfers from other mental hospitals should not enter the admission wards.

(iii) The fæces of all new admissions should be subjected to a bacteriological examination, preferably after a provocative dose of vaccine has been given.

Immunes.—Chronic mental patients constitute a heterogeneous group with diminished susceptibility. The spread of disease can be limited by the division of population into as many self-contained units as possible : the smaller the average ward, and the more independent, the less will be the risk of infection. Ward transfers should be infrequent.

4. *Minimizing Conveyance.*

The conveyance of dysentery in mental hospitals being contrived almost entirely by human beings, the aim of efficient control is to break, at as many points as possible, the carriage of fæces from anus to fingers and mouth.

(i) The first requisite of any mental hospital is lavatory accommodation adequate, in type and number, to the peculiar needs of its population.

(ii) Such a provision must be accompanied by the education of the patient in personal hygiene. He must be trained to regular habit. He must be persuaded to be clean. He must be compelled to wash his hands after using the lavatory, and before each meal. If, despite attempted education, the patient is persistently dirty or faulty, a rigid antiseptic nursing technique

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should be evolved, ranging from the sponging of anus and hands of the patient who is merely careless, to the isolation of degraded patients in specially equipped wards.

(iii) The improvement in personal hygiene should be supported by a rigorous campaign to promote the general cleanliness of the hospital.

(iv) The activities of working patients should be severely restricted. No patient should handle food. No patient should perform sanitary duties. Only selected patients should work in the wards, and under scrupulous supervision.

(v) The hospital building must provide space, light and air.

5. *Measures to Increase Resistance.*

(*a*) *Care of the alimentary tract.*

Food.—The dietary of a mental hospital is largely governed by considerations of cost, and the recommendations of the Committee on Dietaries in mental hospitals have been largely forgotten. Without recapitulating these recommendations, three principles of dieting can be stressed: digestibility, a high protein content, and an adequate vitamin value.

Teeth.—Septic teeth must be eliminated, and where dentures are impossible the diet should be modified.

Constipation.—This should be treated primarily by the exhibition of fluid, adequate exercise, and the regulation of habit. Laxatives, where necessary, should be given daily: practically nothing is more inducive to chronic intestinal catarrh than the large weekly dose of salts.

(*b*) *Specific immunity.*

Choice of antigen.—Absolute specificity is somewhat hypothetical, at least as regards the Flexner bacillus, for even within the sub-groups there are further antigenic variants of a complex nature, governing toxicity and capacity for growth, and modifications dependent on the environment of the organism *in vivo* and *in vitro*. The antigen, therefore, should be prepared from as many strains as possible within the type. Our practice is to keep 6 to 12 strains; as a new strain comes in, we scrap the oldest tube. Further, it is logical to include a high proportion of strains taken from acute cases.

Vaccines are made every 4 months to avoid excessive subculturing. We have rarely to pass our stock through broth, as the supply of new cases has been, until lately, good. The organism is sown on agar, incubated for 48 hours, and washed off in saline.

Killing.—The method of killing the organism should, as far as possible, avoid alteration of the bacillary antigen. The detoxication of the Flexner bacillus, with its feebly soluble toxin, is unnecessary. The killing of the organism by chemical agents—phenol, ether, chloroform, formalin, and the various salts—holds no obvious advantages. The antigenic value, for

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instance, of a formolized vaccine is slight. Nor can we perceive the virtues of "sensitized vaccines". The simplest method is killing by heat at the lowest possible temperature. The Flexner bacillus is easily killed by exposure to 50° C. for 1 hour if the emulsion is well mixed and not too dense. It should be tested for sterility by plating, using several large plates. Very occasionally a further brief exposure to heat is necessary. The emulsion is diluted to the required strength. Only 0·1 % phenol is added as preservative. No vaccine is kept more than 6 months. If mass inoculation is contemplated, if there are no laboratory animals, and the pathologist is tired of acting as such, it is wise to try the effect of a small dose on a few patients.

Combined vaccines.—If specific immunization is adopted for a variety of infectious diseases, or types of one disease, either each antigen may be injected separately, or they may be combined in a single dose. The latter course, from the patient's standpoint, is more humane, but offers certain difficulties.

(*a*) Certain mixtures of bacteria do not form a uniform emulsion, *e.g.* Sonne dysentery and Flexner dysentery.

(*b*) The combined dose must be appreciably greater, or the individual antigenic response appreciably less.

In this hospital we were faced with the problem of four concurrent diseases: Flexner dysentery, Sonne dysentery, typhoid and paratyphoid. Our procedure was this:

(*a*) We did not attempt immunization with Sonne, because, first, it was the least important disease, and second, the emulsion does not mix with others.

(*b*) We used a mixed emulsion of typhoid, paratyphoid and Flexner, varying the proportion of the individual components according to our estimate of the individual activity of the disease. At present this is 1, 2, 2; thus, in the intravenous dose of 20 millions there are 4 million typhoid, 8 million paratyphoid, and 8 million Flexner; in the subcutaneous dose of 2,000 millions the same proportions hold.

Method of Administration.

"*Inoculation.*"—It is usual and customary to administer vaccines subcutaneously or intramuscularly; so much so, in fact, that a clinical habit has become a clinical principle. Vaccines are modified in all sorts of queer ways to allow "inoculation"; and if a vaccine does not produce immunity when administered thus, it is considered to be void of protective power. To our mind the simple "inoculation" of any bacterial product is a poor and uncertain method of obtaining immunity. The antigen of any pathogenic organism is primarily a tissue poison. When "inoculated" a quantity of a very powerful irritant is concentrated in the immediate area of the point of injection. There is a prompt inflammatory (and very painful) response, one object of which is to localize the irritating particles of the damaged tissue. The absorption of antigen, therefore, is slight, delayed and uncertain. The local reticular cells

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may produce a minimal quantity of antibody, but there is no guarantee that any sufficient stimulus is supplied to the remote and more numerous cells of the reticulo-endothelial system. There is no certainty of a generalized immunity, and before that immunity can exist, there must be a long period during which the amount of circulating antigen is too small to stimulate the reticulo-endothelial system, and merely sensitizes other tissues—leaves the body, in fact, more open to infection.

We have employed intramuscular injection on the female side of this hospital (500 patients) for the past 18 months. The heat-killed emulsion of Flexner produces less local disturbance, and less constitutional disturbance (except for some very severe reactions which are inexplicable except as individual idiosyncrasies) than a corresponding emulsion of *B. typhosus*. It is possible, therefore, to employ a larger initial dose. We give 1,000 million to a healthy subject of average weight; the dose is modified according to weight and general condition. A second dose of 2,000 million is given after 7 days, and again every 4 months.

1. The formation of antibodies is variable; there is a lag period of 10 days, a slow and uncertain rise, and a comparatively rapid fall, corresponding to a mild attack of the disease, which affords some, but not complete protection.

During the lag period the subject is more liable to infection; it is therefore bad policy to inoculate subcutaneously patients in an infected ward; by so doing we caused a mild epidemic.

It is probable that maximum protection is afforded just after the maximum rise; the saturation of the tissues with antibody is slight; and the protective effect transient. The injections of vaccine must therefore be frequent, and continued over a long period.

2. There has been a fall in the number of cases; in 18 months there have been only 3 cases, against an average expectation of 15 to 30.

3. Two cases were very mild; the last was very severe.

Oral and rectal vaccines.—In point of historical fact oral immunization preceded inoculation, although its revival in practice, accompanied by the theory of "local immunity", is of comparatively recent date. The theory of local immunity as propounded by its chief advocate is, briefly, that different organisms have an affinity or predilection for different tissues, and that by saturating the particular tissue with specific antigen the receptivity of that tissue is exhausted, and infection is thus made impossible. Specific immunization, therefore, should follow the probable route of infection. This route, in dysentery, is by absorption from the alimentary tract.

Our results have been disappointing. We selected two wards (134 patients) in which dysentery was active, and gave to each patient 3 doses of heat-killed vaccine, each of 100,000 million, according to the prescribed technique, on a fasting stomach and preceded by a dose of bile. This procedure was repeated after 3 months. There was no diminution in the number of cases, or in their

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severity, either relative to the previous 6 months, or to other infected wards, not immunized.

When giving Flexner vaccine per os we tested the blood of six patients from the second day onwards without demonstrating any agglutinins, except those present in some patients before administration. A fortnight after administration the blood serum of one patient was highly bactericidal, and the residual agglutinins had disappeared :

14.iv.33 : Agglutinins 25.			
28.iv.33 : ,, 0.			
Bactericidal test :	Control	784	100
	Serum diluted $\frac{1}{10}$	47	6
	,, ,, $\frac{1}{5}$	22	3

Intravenous vaccine.—In theory at least the route of choice would appear to be the intravenous. The antigen is widely and immediately distributed throughout the blood-stream, and there is a certainty that a high proportion will be absorbed by the reticular cells of the spleen, liver and bone-marrow. The maximum response of antibody is obtained with the minimum dose of antigen. The response is prompt ; the concentration of antibody is maintained in the blood over a long period, and presumably the tissues are endowed with a corresponding degree of protection against the disease.

In practice intravenous inoculation is just as simple as subcutaneous inoculation. It takes little more time in execution ; it is less painful ; there is no focal reaction ; there is less risk of sepsis ; and the systemic reaction can be exactly calculated.

We have used this method on the male side of this hospital (330 patients) for the past year. An initial dose of 20 millions is followed in a week by a dose of 40 millions repeated every 6 months.

1. The formation of antibodies is constant. There is an almost immediate rise of agglutinins, rising to a figure 10 or 20 times greater than that produced by intramuscular inoculation, and lasting longer. The production of antibody is, in fact, much greater than that produced by the actual disease. It is possible that the peak may be above the optimum figure of antibody concentration.

2. There has been a fall in the number of cases. The apparent fall is slight—from an expectation of 15 to an actuality of 6. The real fall is probably far greater. Intravenous inoculation lights up any latent disease, and the majority of cases recorded are certainly not new ; they have a previous history of diarrhœa, and they occur immediately following inoculation. The real fall is, probably, from 15 to 2.

3. All the cases recorded were very mild.

We have also successfully experimented with other vaccines given intravenously. Autogenous intravenous vaccines yield good results, without any

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focal or systemic reaction in staphylococcal furunculosis, and *B. coli* pyelitis. The complete absence of focal reaction is very notable. For instance, in a very advanced case of tuberculosis (lungs and kidneys) a millionth of tuberculin A.R. given subcutaneously produced a very marked focal reaction, with exacerbation of general symptoms; ten times this dose intravenously was without appreciable effect; after four injections the patient could tolerate a hundredth milligramme.

On the other hand, where there is an avascular and very chronic lesion, as in the cholecystitis of the typhoid carrier, intravenous vaccine sends the somatic agglutinins rocking, but does not touch the excretion of the germ.

Mass immunization.—Whichever method of administration is adopted, and whatever antigen is employed, it is essential that each member of the community be equally immunized. There must be no exceptions. If the patient is ill the dose is decreased or delayed, not omitted. The only danger of immunization is that it may not be sufficiently thorough, either for the individual or the mass. Half-hearted immunization will simply increase the ratio of undiagnosed cases, and render effective treatment and segregation impossible.

It is especially necessary to immunize the most susceptible fraction of the population, the new admissions. Abolish susceptibility, and the disease must die. The inoculation of the "older" population is relatively unimportant; they are, relatively, insusceptible.

Finally, in a recently infected ward, unless the active excretors have been diagnosed, the only safe protective agent is an immune serum; intravenous inoculation is relatively safe; subcutaneous inoculation should not be employed until the active excretors have been isolated.

The value of specific immunization.—The degree of protection afforded is considerable, both in minimizing the individual severity of the case and of the number of cases, but it is not complete. To take a very recent example. A ward of 50 degraded female patients remained free from dysentery for a year. During that year each patient received 4 intramuscular injections of vaccine, each of 2,000 million. A solitary case of dysentery occurred. The ward was searched for excretors, without result. As a further precaution, the patients were inoculated intravenously, resulting in an average titre of over 100 R.T. A month later there was another case of dysentery. Within two months there were six, until at last the infector—a patient excreting very intermittently—was discovered, and the disease was checked. Now the infected patients possessed, apparently, a surplus of specific antibody, and the type of the infecting organism was identical, according to cross-agglutination, with the organism from which the vaccine was prepared!

Conclusion: Efficient Control.

On paper, the scheme of control suggested may appear somewhat formidable. The practical administrator of a mental hospital, viewing dysentery as only one of many infective diseases, and infective disease, in bulk, as only a minor

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part of the whole administrative problem, will refuse to be intimidated by the formidable picture of the disease which we have drawn: he adopts the time-honoured principle of expediency. If the incidence of unmistakable cases rises, he segregates one, and notifies the other. If, perchance, there is an epidemic (very mild), he remembers that dysentery is a sanitary index, digs out a bad drain, and sends his drinking-water for analysis. The analyst's report (as such reports often are) is non-committal and indefinite, so that the expedient administrator, when the second epidemic comes, consults a pathologist, who prepares a vaccine, and stabs various groups of patients. When the third epidemic comes, he builds his own laboratory, and dabbles mildly in bacteriological diagnosis. He is surprised to find so few positives; but in the fourth epidemic he discovers a real "carrier" and descends in wrath upon the wards, with stringent orders that dysentery bacilli shall be confined to their proper place. And when those recalcitrant little pests refuse to do anything of the sort, and proceed to a fifth outbreak of epidemic disease, the expedient administrator asks the taxpayer for a new hospital.

The moral of this fairy-tale is that mere expediency is not enough; only a deliberate and systematized method of control can hope to eliminate endemic bacillary dysentery from a mental hospital. No single measure will suffice. The laboratory, by itself, is quite unable to check the disease. There must be knowledge of the disease-process. There must be a deliberate assessment of the endemic level of the disease. There must be prompt and accurate diagnosis, and a reliable test of cure. In those matters the laboratory worker is indispensable, though not by any means infallible; he is, shall we say, a competent observer and critic of events. The practical attack on the disease is in the wards and in the person of the patient. Diagnosis without efficient segregation is useless. Immunization is only complementary to segregation. The root-problem is that of conveyance: how can the standard of personal hygiene throughout the hospital be raised to such a pitch that fæcal contamination is impossible?

Working from the narrow angle of the laboratory, we ourselves are afflicted by a sense of complete failure. After four years' work, we seem to know a little more about the disease, and the incidence figures seem to be falling. Someone has said: "If one can find an institution in which dysentery is at a lower ebb than in some other institution where the environmental conditions are better, it does not follow that what has been done in the second institution was futile. . . . The Mills of God grind slowly." They do.

Cases.

The total number of cases observed was 87. Eight of these came to the post-mortem table.

Only those cases are quoted which illustrate the text. Few cases are as

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complete as we would like them to be, for reasons obvious to anyone with a knowledge of mental hospital routine.

The post-mortems are similarly disappointing; bacteriological examination of material is vitiated by the fact that the post-mortem was usually delayed until 36 hours after death, by which time the only organism likely to be recovered, if it exists, is *B. typhosus* or *paratyphosus B* from the bile.

E.M—, male. *A Sonne carrier.*

Admitted 25.ix.33. Fæces positive to *B. Sonne* on admission. Serum agglutinated *Sonne* at 1/25. No symptoms of dysentery. Died of intercurrent disease 10.vii.35. No p.m.

H.S—, male. *Chronic dysentery; undiagnosed transfer.*

Transferred from another mental hospital 25.i.25. On admission fæces negative; serum agglutinated *Flexner* at 1/25. Passed blood and mucus. Fæces positive to *Flexner Z* 31.i.35, negative 2.i.35. Serum on 7.ii.35 and 14.ii.35. Continued to agglutinate *Flexner Z* at the same dilution. Diagnosis: Chronic dysentery; not a recent case, and not infected in this hospital. Differentiation of chronicity only made possible by Widal.

O. S—, male. *Chronic dysentery; working patient.*

Symptoms of acute dysentery 16.ii.32; fæces positive to *Flexner Z*. Periodically suffered thereafter from occasional diarrhœa; 7 consecutive examinations of fæces negative.

A worker in the garden, who often distributed fruit and vegetables, and therefore suspect. Sigmoidoscope examination 26.vi.34: Mucosa unhealthy, with typical granulations and one small, but definite ulcer. Sigmoid swab: Douglas and Colebrook No. 8.

27.iv.34: Fæces negative; mucus negative; muco-pus gave *B. Flexner Z*. Since then 4 negative fæces. Blood-agglutination constant at 1/25. Diagnosis: A very intermittent excretor. Case illustrated: (1) The intermittent excretor; (2) the danger of the working patient; (3) symbiosis of *Flexner* and D. and C. No. 8; (4) value of the sigmoidoscope.

J. H—, male. *Chronic dysentery.*

Acute dysentery 1.viii.31; fæces positive to *Flexner Z*. Thereafter 4 negatives. Fæces positive again 19.v.32; thereafter 9 negatives. Fæces again positive 13.ix.33; thereafter 7 negatives. 12.v.34: D. and C. No. 8 recovered from a loose stool; thereafter 2 negatives. 11.ii.35: Following an intravenous injection of vaccine, fæces positive to *Flexner*. Case illustrates: (1) Intermittent excretion; (2) value of intravenous vaccine in lighting up latent disease; (3) interruption of D. and C. No. 8 into a *Flexner* series.

E. B—, male. *Chronic dysentery.*

First positive with symptoms, 14.x.30, *B. Flexner*; thereafter 4 negatives. Positive again 15.xii.33; thereafter 1 negative.

Sigmoidoscopy 24.ii.34: Mucosa thickened, dark and congested. Several ulcers, one large. Floor of ulcer scraped; 2 swabs yielded D. and C. No. 8.

On 5.iv.34 fæces contained mucus; D. and C. No. 8 isolated. 16.iv.34: Fæces negative. 18.iv.34: Fæces again showed D. and C. No. 8. On 24.iv.34 the fæces (ward specimen) were negative, but mucus gave D. and C. No. 8. On 7.i.35, following intravenous vaccine, *Flexner* was again recovered. Blood-agglutination steady at 1/25. Case illustrates: (1) The subacute case; (2) value of sigmoidoscopy; (3) potential pathogenicity of D. and C. No. 8.

F. C. H—, female, æt. 63. *Reaction to vaccine.*

Routine inoculation with dysentery vaccine (2000 mill. heat killed *Flexner*) 19.iii.35. Died of coronary thrombosis 20.iii.35.

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Post-mortem 21.iii.35: The intestine was congested, particularly in the lower ileum, and the mesenteric glands were swollen. There was considerable mucous exudate.

E. C—, female, æt. 80. *Acute lesions.*

Admitted 1923. No history of diarrhœa. Four negative fæces. Contracted acute dysentery 14.v.34. Fæces yielded *B. Flexner* in almost pure culture on 15.v.34 and 16.v.34. Died 28.v.34.

Post-mortem 30.v.34: The whole of the gut from stomach to rectum was inflamed, with numerous small ulcers in the small and large intestine of recent origin; there was considerable sloughing, hæmorrhage, and a muco-purulent exudate. The liver and spleen were enlarged, and there were inflamed mesenteric lymph-vessels and glands. Microscopic sections of the liver and spleen showed intense congestion, with focal necrosis. It is only fair to note that the only organism recovered at the post-mortem was *B. paratyphosus B* from the bile. The patient was a carrier of this organism, and the acute lesions were dysenteric.

M. S—, female, æt. 40. *Acute lesions.*

Suffering from large gluteal abscess. Contracted dysentery 27.v.34; fæces on that date gave almost pure culture of *B. Flexner*. Died 29.v.34.

Post-mortem twelve hours later: Mucous membrane of large intestine and of small intestine irregularly thickened, with patches of dusky congestion. The lower colon was least affected. The only definite ulcers were in the ileum; these were of recent origin. The mesenteric glands were swollen. The spleen was soft and pulpy. Bacteriological examination: The blood-stained exudate yielded *B. Flexner*; the bile was sterile.

A. F—, female. *Chronic dysentery.*

Found excreting *B. Flexner* without symptoms 11.ix.32. Died 18.xii.33.

Post-mortem 19.xii.33: There was slight congestion and catarrh of the ascending colon, and similar changes in the lower ileum, where the lymphoid aggregations were swollen; near the ileo-cæcal junction there were two small ulcers. The lymphoid aggregations of the lower ileum were swollen; near the ileo-cæcal junction there were two small indurated ulcers. There were a few enlarged mesenteric glands. Bacteriological examination of the bile, intestine and glands negative.

L. P. W—, female, æt. 58. *Chronic dysentery.*

Fæces positive, with symptoms, 8.viii.32. Symptoms recurred on various occasions, but fæces invariably (6 examinations) negative. Last negative 1.vi.34. Died 5.vi.35.

Post-mortem 6.vi.35: Pus-pits and mucous cysts in large intestine; small intestine inflamed and congested, and coated with a muco-purulent exudate. Bacteriological examination: Bile and intestine, no N.L.F.

E. F. B—, female, æt. 58. *Chronic dysentery.*

History of intermittent diarrhœa with excretion of mucus. Three negative fæces. 31.i.34: D. and C. No. 8. 7.ii.34: Found excreting *B. Flexner* without symptoms. Died 20.iv.34.

Post-mortem 22.iv.34: There were many pus-pits and a few mucous cysts in the descending colon. The small intestine was healthy. Bacteriological examination of gall-bladder negative.

M. A. K—, female. *Transient excretor.*

While resident in infected ward found excreting *Flexner* without signs or symptoms, 29.iii.34. Blood agglutination 1/250. No previous history of diarrhœa. Scrupulously clean. Negative sigmoidoscopy. Ten consecutive stools free from mucus. Fæces, after 29.iii.34, persistently negative.

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E. H—, female, æt. 62. *Transient excretor.*

Found excreting *Flexner* without symptoms 24.vii.29. Thereafter fæces consistently negative (over 20 specimens). Labelled a carrier (our mistake) in 1932, when dysentery broke out in the ward where she was resident.

Post-mortem 3.iii.33: Intestine absolutely healthy. A transient excreter, falsely labelled a carrier. Gall-bladder and intestine: No. N.L.F.

A. R—, male. *Chronic dysentery. In hospital stores.*

Worked in main stores, handling food. A single loose stool observed by a vigilant night-nurse, and a specimen sent to the laboratory. Fæces positive to *B. Flexner* 15.xii.34. Blood agglutinations: 20.xii.34, 1/25; 2.i.35, 1/25. An intermittent excretor.

M. E. G—, female, æt. 82. *Subacute dysentery.*

Admitted 1.ii.33. Exposed to infection. History of diarrhœa, with blood and mucus. *B. Flexner* recovered from stool on 5.iv.34. Treated vigorously with sod. sulph. and died on 18.iv.34.

Post-mortem 19.iv.34: The whole large intestine and the terminal 5 in. of the ileum were intensely congested and œdematous. There were a few very chronic ulcers in the descending and some pus-pits in the pelvic colon. An acute exacerbation of a chronic case. Gall-bladder and intestine: No N.L.F.

R. D—, male, æt. 80. *Chronic dysentery with negative fæces.*

Acute dysentery with positive fæces 18.ix.33. Apparently cured. No further symptoms. Six negative fæces. Died 17.ix.35.

Post-mortem 18.ix.35: Granulations in ascending colon; small mucous cysts and two small polypi in sigmoid colon. Probably chronic dysentery.

A. S—, male. *Chronic dysentery. A farm worker.*

A more or less independent worker on the farm. Previous and very definite history of diarrhœa. A week following intravenous vaccine, on 18.iv.35, he had an attack of diarrhœa, with mucus in the stool; fæces positive to *B. Flexner*. No further investigation. Isolated for a while, but now again working on farm. Probably an intermittent excretor.

A. T. P—, male. *Combined infection.*

Sonne: 24.x.33. *Flexner*: 30.i.35. Excreted *Flexner* following intravenous vaccine. Probably a *Flexner* carrier.

M. L. S—, female. *Severe reaction to vaccine.*

A new admission. Collapsed two hours after a first injection of 1,000 millions. She produced a series of rigors; that same evening the temperature rose to 106·2 F. The patient was very ill for a fortnight. Certain possibilities can be dismissed: (1) There were no signs of sepsis and the blood-culture was sterile. (2) She was not the subject of dysentery; the fæces and Widal were negative on admission. (3) The needle did not enter a vein; the maximum agglutination titre was delayed and comparatively low.

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HYPNOSIS IN MENTAL HOSPITAL PRACTICE.

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THE Mental Treatment Act of 1930 has brought within the purview of the mental hospital that large class of psychoneurotic and "early psychotic" patients who formerly drifted despairingly in the wilderness between orthodox medicine and the quack. The purpose of this paper is to show how an attempt is being made in this hospital to deal with this heterogeneous class of patients by means of psychotherapy, carried out by the ordinary medical staff of the hospital, without interference with their routine duties.

Since Cefn Coed Hospital opened in 1932 a large number of voluntary patients have presented themselves for treatment. In the year 1935 there were 72% of voluntary patients out of 276 admissions. It is obvious that this supply was largely drawn from a type of patient new to mental hospital practice. The medical officers of an ordinary mental hospital then found themselves confronted with diagnostic, prognostic and therapeutic problems such as had not come within their experience prior to the Act of 1930.

In many such cases the ordinary routine treatments of diet, drugs and such special measures as occupational therapy, narcosis and onslaughts on septic foci were attended by relatively scanty success. In addition the full histories which were obtainable in this hospital, and the results of prolonged, careful, clinical observation, brought into striking prominence the importance of a psychogenic factor in many of our cases.

Accordingly it was essential, in the interests of these patients, to make a somewhat more extended attempt at psychotherapy than, we believe, is usual in the ordinary mental hospital. The large number of patients requiring such treatment rendered a prolonged and exhaustive psychological analysis of each case impracticable. An efficient short cut both to mental exploration and subsequent adjustment appeared to lie in the use of hypnosis. As McDougall puts it:

"The value of direct suggestion for relief of symptoms in simple cases should not, I think, be denied or ignored. But the chief value of hypnosis and of hypnotic

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suggestion lies, to my mind, in their use as aids to exploration and the relief of amnesia, both repressive and dissociative. The use of hypnosis offers a short cut in the stage of exploration, which, in many cases, for lack of such aid, may be very prolonged. . . . And Prof. Freud himself has acknowledged its wide usefulness in such cases . . . etc."

And again:

"I believe in the value of mental exploration as deep as the case requires; and I regard free association and dream analysis as important methods of exploration. But I also hold that exploration in hypnosis is, in many cases, useful and entirely justifiable."

Using hypnosis as such a short cut, it was decided to test its applicability to the new types of cases now being admitted.

At this point we may usefully indicate a variety of indirect factors which greatly favoured this work. In the first place the compactness of the Borough of Swansea greatly facilitated investigations of the various environmental factors bearing on the patient's illness. Practically every new patient is visited twice a week by his relatives. This close contact with the relatives offered numerous opportunities for adjusting the environment as required. In the second place the services of a trained social worker enabled us to obtain full histories, and to direct the care of our patients after leaving hospital. Before taking the social history the social worker was able to make a careful study of the case-notes to date, and to work in the closest co-operation with us.

It may seem strange, but it is nevertheless true, that among the people of Swansea Cefn Coed Hospital is not regarded as a hospital for "mental" patients as the word is commonly understood. The admission block, into which all new cases are received, is some distance from the main building, and in many important respects, including that of "atmosphere", resembles more an ordinary hospital than the accepted idea of an institution for the mentally ill. We are lucky also in having an admirable team-spirit among the medical staff, resulting in free interchange both of ideas and patients. Clinical meetings of the whole medical staff are held weekly, at which diagnosis is discussed, the progress of patients checked, and lines of treatment decided upon and reviewed from time to time.

The work was started in March, 1936, and proceeded on the following approximate plan. First the case was thoroughly investigated, physically and mentally, along the ordinary lines. Then an attempt was made to assess the factors which had given rise to the patient's maladaptation. Any physical causes were treated according to medical and surgical principles. The social history was carefully scrutinized and correlated with the clinical findings. Where psychogenic factors were suspected, the patient's susceptibility to hypnosis was tested by an attempt to induce this state. In addition to cases of obvious psychoneurosis, we tried those cases of so-called psychosis who were

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not making satisfactory progress, but whose illness seemed to have a psychogenic basis.

The following are the summaries of twenty cases, representative of the series we are attempting to treat in this way. In some of them hypnosis could not be induced, even after repeated attempts by both of us. Failure, for example, occurred, by reason of their inability to co-operate, in most of the psychotic patients we tried to hypnotize.

This agrees with many previous statements to the effect that cases diagnosed as psychoses are not amenable to hypnosis. In many patients hypnosis was attempted, successfully or unsuccessfully, by both of us. In all of them views were mutually expressed and discussed concerning the details of treatment and the surmounting of difficulties as they arose.

CASE 1.—This case illustrates the use of hypnosis for implanting a required suggestion :

Female, æt. 34. Cashier. Treated as an out-patient.

She had previously been in this hospital for six months, and had left totally unimproved in a state of despair.

She had been admitted with a previous history of three years' duration of increasing sleeplessness and a peculiar obsession. She thought that £1 notes came down her sleeves, were in her stocking, were in the paper she laid the fire with, so that she had to rake over the ashes to reassure herself. She had a team of other patients examining her slippers, shoes and other clothes. She frequently dreamt that she was enclosed within a high wall which she could not climb. As the result of all this she had become irresolute, anxious and full of doubts and fears of the future, as she was incapacitated from handling any money whatsoever.

At this time one of us (E. H. K.), who had previously studied hypnosis, was asked to attempt this procedure on account of the complete failure of all ordinary methods. She was accordingly sent for again, and interviewed as an out-patient. Hypnosis was easily induced. She passed readily into the deepest stage. Negative hallucinations were easily suggested and she carried out post-hypnotic suggestions. During her previous stay in hospital a correct formulation of the causes of her illness had been presented to her by the other author (C. L. C.). She had a domineering mother, on account of whom she had sacrificed for ten years an exceptionally promising career as hotel cashier ; also she was sexually maladjusted. The genesis of each symptom had been laboriously worked out, explained to her and accepted by her intellectually but her symptoms had persisted.

In hypnosis all her symptoms were removed permanently at one sitting of two hours' duration by counter-suggestions. She was told in hypnosis that her obsessions would cease completely. Money was placed in her hands and she was ordered to count it. While doing so she was awakened, and found that she could continue to count it without experiencing any of her former fears.

As a matter of interest two further sessions took place, at which the previous formulation of her condition was checked up and found correct. She recovered completely, and was able to resume a normal life without hindrance by symptoms.

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CASE 2.—This case illustrates the great value of hypnosis as a quick method of bridging amnesic gaps :

Female, æt. 22. Voluntary patient. Children's nurse.

She was admitted on February 22, 1936, in a confused, dreamy state, saying that she was " all mixed up ". " I feel in an awful muddle. I can't remember when I first met my young man. I am mixing up the names and places and numbers. I feel I have been in a fog for a very long time." In a day or two she settled down and showed herself to be very anxious to co-operate. She could give an account of all events in her life up to six months previously. Her history since then showed the presence of numerous extensive gaps, of which she had only the most fragmentary memories. Her account of all this period gave the impression of phantasy, as she mentioned names and events totally discrepant with any history that could be obtained about her. Just prior to admission she had been involved in a minor car accident. It was only then that she realized that something was wrong with her and sought treatment.

Hypnosis was induced with remarkable ease. She passed into the deepest stage in which hallucinations could be suggested. Mental exploration in this state spread over several days revealed the presence of a series of extensive fugues almost constituting a continuous fugue for the whole six months. In her waking state she was quite ignorant of these events, but in hypnosis, although she lived through the events of the fugues, she retained her personality. In other words there had been an extensive dissociation occurring at the highest psychic level. For example, she found a house-key in her handbag which she could not account for, and she gave the number of her house as 44 instead of 2. In hypnosis it was found that the key belonged to a house numbered 44 in another town. An adequate emotional situation was revealed as the cause of the original dissociation. She was very closely attached to her father, who was dying of cancer, and was engaged to an Italian in London, of whom her father disapproved. While worrying over these troubles she became innocently entangled with two other men in Swansea, one of whom had a house numbered 44 and had the car in which the accident occurred. During her fugues she alternated between the latter two men, who were unaware of each other's existence, and neither of whom knew of the father or of the Italian.

The gaps in her memory were easily filled up, the dissociation was bridged and re-integration effected.

It is impossible adequately to condense this case. It contains numerous features of general interest, is extremely long and detailed, and it is hoped that it will be published separately in full. The result is, up to the present, complete recovery, and the patient is leading a happy, useful life.

CASE 3.—This case, regarded as a psychoneurotic, shows that hypnosis cannot always be induced in such cases :

Male, æt. 28. Voluntary patient. Lorry driver.

Admitted complaining of loss of confidence, insomnia, headaches and a left foot-drop (functional) which prevented him from driving his lorry. He was afraid he was losing his memory, and complained of blank feelings. He had obsessional visions of running over people with his lorry, but he had never

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had an accident in twelve years of driving. He was regarded provisionally as a case of anxiety state. The supposed causes included a number of assaults on his pride in the past two years, and the recent death of his mother. He had undoubtedly been spoiled by his family. There were no apparent conflicts over sexual affairs. Extended investigations failed to establish how his lorry-driving came to be affected, and word-association tests shed no light on his case.

Hypnosis was attempted without any success on several occasions; he appeared to be genuinely non-suggestible. He left hospital relieved, his foot-drop having recovered, and he became able to drive again and was much less anxious.

Three months later he presented himself at the out-patient clinic completely relapsed. Further repeated attempts at hypnosis failed. He refused to return to hospital, was not certifiable and was lost sight of. Two months later, unfortunately, he committed suicide.

CASE 4.—A true melancholic; hypnosis attempted to test its applicability in such a case:

Female, æt. 48. Housewife. Married. Voluntary patient.

She was a straightforward case of recent melancholia who complained of lack of confidence, laboured under a feeling of unreality and despaired of ever recovering. She had made no progress for a year. Hypnosis was attempted in order to try to give suggestions of confidence, but she was entirely unsuitable. She has since recovered sufficiently to leave hospital relieved.

CASE 5.—A case in which hypnosis revealed unexpected amnesias, exposing the true nature of the illness:

Male, æt. 26. Collector. Voluntary patient.

He was admitted with the following history. During the past five years he had run away from home nine times, and on the last occasion staged an elaborate suicidal attempt in a London hotel. He said this had happened because his relatives were unduly critical of him. He worked in his father's coal office together with an elder sister, whose superior attitude he bitterly resented. In addition he disliked the coal business, but had made no efforts to seek more congenial employment.

It was noticed that he showed a symbolic hand-washing ritual, but he did not complain of this or appear to notice it himself.

He was readily plunged into deep hypnosis, and the information gained in this state enabled definitely correct advice (as events subsequently proved) to be given to his parents. In hypnosis a series of incestuous episodes with his three sisters, starting when he was 18 years old, came to light. As each grew up and objected he took on the younger one. The hand-washing was traced to its commencement at the episode with the first sister, and symbolized a repressed sense of guilt. The elder sister had become aware of his attentions to the younger ones, producing a singularly unfortunate situation for him in his employment.

He accepted all these explanations. His father wished him to continue to take over his business, but in view of the knowledge gained in hypnosis, he was categorically advised that it was imperative that this should not happen. The patient left hospital satisfactorily adjusted.

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CASE 6.—Although hypnosis could not be induced in this man, the suggestive effect of the technique was beneficial :

Male, æt. 48. Voluntary patient. Bus conductor.

This case superficially resembles Case 1. He was admitted in an intensely agitated state, complaining of insomnia, buzzing in his head, and obsessive ideas. "It feels as if my thoughts are pulled out through the left side of my head." He also had compulsive ideas ; he had an irresistible impulse to pick up small pieces of paper on the road similar to bus tickets associated with the words "Spirit of God", running in his head. He had become unable to ride on a bus. He was a lay preacher and of good intelligence. He had considerable insight and endeavoured to co-operate fully in hypnotic treatment. He could never be hypnotized. He became a little drowsy, and his letters home revealed that he had experienced marked subjective benefit from the suggestions given to him.

The bus company in which he had previously been an inspector had been bought up by a larger company, who insisted on supplying their own inspectors. He had perforce to accept a position as conductor. The blow to his self-regard occasioned by this "degradation" was the cause of his attack. He left hospital greatly improved and shortly afterwards recovered completely, and is now happily filling his duties as a conductor.

CASE 7.—A case of obsessional neurosis totally uninfluenced by attempts at hypnosis :

Female, æt. 34. Housewife. Voluntary patient.

This was a patient who suffered from pulmonary tuberculosis with tubercle bacilli in the sputum. She complained of obsessive ideas and phobias, e.g., that she infected everyone with tuberculosis. She was afraid of a "thought sticking", and of upsetting the sputum mug. She had become a maze of complicated rituals ; the act of expectoration, which was frequent, took five minutes. Washing her hands took half an hour. She was constantly dogged by the fear that she was not conscientious enough over these rituals. She was subject to compulsive utterances, e.g., "Did I cough ?" interspersed freely in every conversation.

She was a case of obsessive and compulsive psychoneurosis. Hypnotic exploration was attempted on at least seven occasions, entirely without success, although she showed numerous suggestible phenomena. Prolonged analysis in the waking state was carried on for nine months. The causes of her symptoms were each unravelled to the last detail. She refused to accept them, and preferred to remain a neurotic rather than face up to her conflicts. She is still in hospital, appearing to enjoy her disabilities.

CASE 8.—Illustrating a use of hypnosis in alcoholism :

Male, æt. 36. Voluntary patient. Salesman.

This man was admitted in a state of delirium tremens. Within a week this had yielded to the usual measures, and a history of chronic alcoholism dating from 1927 was obtained. He was unable in the waking state clearly to discuss the causes of his addiction. He was ashamed to discuss his personal affairs, and it was decided to investigate in hypnosis.

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He proved a good subject, though the onset of full hypnosis was always slow and tedious, and complete success was only attained at the third attempt. In hypnosis the following facts emerged : He was spoilt as a child. He entered the army when 16, and had a good time as batman, but was disillusioned when he reached the trenches. He was blown up on the eve of " going over the top ", and sustained a recurrent dislocation of his shoulder, requiring successive muscle-grafts. In addition he had a hysterical aphonia lasting six weeks.

At this time he drank but little. In 1927 he was appointed to a responsible salesman's post, and acquired the added responsibility of buying a house and furniture on instalments. From then his drinking rapidly increased. In spite of this his business ability remained good, but his conduct at home deteriorated, and he had beaten his wife on numerous occasions.

These facts were consciously accepted by him and he was then treated by endeavours to strengthen his sentiment of self-regard by giving him suggestions of confidence, ability to overcome his weakness and dislike of the taste of alcohol.

He appeared to respond excellently, and soon felt so well that he insisted on leaving hospital to return to work. He had only been in six weeks.

We felt that the prognosis was bad as (*a*) he had been a spoilt child ; (*b*) his occupation gave him immense temptations to drink ; and (*c*) his stay in hospital had been so brief. Nevertheless, as he was a voluntary patient, he left.

Seven months later he returned, worse than before. In hypnosis he admitted that he had been drinking continuously since the day he left hospital. A deeper analysis is now in progress, and he has promised to remain for six months. Nevertheless the prognosis appears to be almost hopeless.

Case 9.—Here the failure of hypnosis helped to distinguish between psychoneurotic and melancholic depression :

Female, æt. 33. Teacher. Voluntary patient.

She was admitted in a state of agitated depression and was preoccupied with ideas of sin and inferiority. She had a markedly religiose trend of thought, but asserted that the devil had possessed her. She had become very sensitive about superfluous hair on her chin, and continually hid her face in her hands. This prevented her from teaching at school. She asserted, " It was Satan's suggestion. I was not woman enough to master myself ".

She also complained that when out walking by herself she felt she was being pushed against the hedge.

Her father had died during her infancy ; her mother sacrificed much to make her a teacher—an occupation for which she now felt she was unfitted. Just prior to the present attack she had refused an offer of marriage by an eligible clergyman, saying that she felt she was unworthy. The regret engendered by this lost opportunity seemed to us to play an important part in determining her symptom-complex, and at a staff meeting it was considered that she might be a case of hysterical insanity, particularly as for a week she meowed like a cat and later baa-ed like a sheep.

Acting on these considerations hypnosis was attempted. She proved completely unsuitable ; her attention could not be obtained and she believed that some malign influence was being applied to her. Subsequent observation and developments established the diagnosis of agitated melancholia, and she has remained depressed, expressing delusions of wickedness and quite unable

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to carry on her occupation. Her family insisted on removing her from hospital against advice after she had been a patient for six months, as they could not afford further payments.

CASE 10.—A hysterical dissociation rapidly and completely resolved in hypnosis :

Female, æt. 22. Housekeeper. Voluntary patient.

This case and the next are conveniently cited together, as they were sisters. This patient was correctly diagnosed on admission as hysterical dissociation of the personality, and her sister (Case 11), who was admitted a few days later, was at first thought also to be suffering from the same disorder.

The patient was admitted complaining that she felt that she had been changed into a Chinaman, that a Chinaman had entered her body sexually, and his spirit had taken possession of her. Her conversation was rambling and incoherent. At times she spoke in pidgin-English and often used the words "Zacca" and "Wu". In a day or so she became more composed and gave the following history :

She had recently become interested in spiritualism, and her brother-in-law (the husband of the next case) posed as a medium. For a long time she had felt herself to be strongly sexed and, although she had not permitted intercourse, had masturbated freely, with some sense of guilt.

A few days prior to admission she had felt a "sexual feeling" come over her, and had felt as though a Chinaman had taken possession of her body and her face was changing into that of a Chinaman. She had gone to her brother-in-law, who attempted to exorcise what he called "the spirit" unsuccessfully. She became frightened and her family sent for the doctor, who advised admission to hospital. She had no conscious associations with "Chinaman", "Wu", or "Zacca".

She was hypnotized with ease, passing into the deepest stage. She recalled how, when cooking in London six years previously, she had been frightened by a Chinaman who had proposed to her. She had recently been reading Lawrence's *Seven Pillars of Wisdom*, in which the word "Zacca" occurs. She also admitted that she had failed satisfactorily to sublimate her sexual instinct. After five sessions she clearly understood all the events which had occurred, and herself stated that her sexual maladjustments were the cause of her trouble.

With this realization her symptoms ceased, she became quiet and no longer confused, and rapidly returned to normal. She left hospital and has remained well for the last six months.

CASE 11.—A case at first immune and later susceptible to hypnosis. The susceptibility influenced the diagnosis in favour of a psychoneurosis :

Female, æt. 24. Certified patient. Home duties. Married.

This patient, the sister of the previous one, was admitted four days later in a state of acute excitement, having thrown herself out of an upper floor window of her house and broken her leg. She said she had come under the control of the "Chinese power", which had been passed on to her from her sister. This "Chinese power" compelled her to shout and to try to throttle her dog and her husband ; it also caused her to inflict enormous bruises on herself, and eventually to throw herself out of the window.

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Owing to our knowledge of the condition of her sister (cited above) immediate and repeated attempts at hypnosis were made, with complete failure, as she was constantly distracted by acute visual hallucinations in which she saw the " power " and her dog.

For a month she remained in this state of turbulent violence, and then gradually quietened. For three months she remained convinced that she was under the control of her sister's " Chinese power ". She declared dogmatically that she felt it coming in through her fingers. At times she impulsively struck other patients in response to auditory hallucinations. She appeared to be a case of paraphrenia with a bad prognosis, though the circumstances attending the onset and her age were against this. Gradually the delusional ideas became less fixed, the hallucinations disappeared and her conduct improved. Quite suddenly she announced that she had recovered. She had no memory for the events of her acute illness. She was now found to be hypnotizable. In hypnosis the psychogenic nature of her illness was revealed as follows :

She was an unwilling participator in her husband's spiritualistic activities. She became intensely jealous of the above sister, who had fallen in love with her husband and had asked him to treat her. Her father then blamed her and her husband for this sister's breakdown, and she was torn between love for her father and love and loyalty to her husband. She had herself endeavoured to exorcise her sister's " spirit ". This sitation had precipitated her attack of acute mental illness and coloured the whole picture. She would appear to be a case of purely psychogenetic psychosis. She made a rapid recovery, and left hospital completely well.

CASE 12.—Another case in which susceptibility to hypnosis confirmed the diagnosis of a psychoneurosis :

Female, æt. 21. Single. Voluntary patient. Home duties.

This girl was a high-grade mental defective, showing mild depression and anxiety for no ascertainable reason. After four months of hospital regime she left apparently recovered. In two days she returned in a state of stupor resembling catatonia. The sudden onset and her previous history suggested hysteria, and this was confirmed by her recovery from the stupor in two days.

Hypnosis proved successful and further confirmed the diagnosis of hysteria, as our experience is that schizophrenics are incapable of being hypnotized. She had not sufficient intelligence to co-operate with detailed mental exploration and no new information came to light. We would suggest that the stupor was a reaction to the shock of leaving the sheltered life of the institution too suddenly.

This time she was given graduated periods of leave from hospital, and in three months was able to be discharged again and has remained well.

CASE 13.—Showing the insusceptibility of a schizophrenic to hypnosis :

Female, æt. 27. Housewife. Voluntary patient.

This patient was obsessed by the word " worry ", which constantly recurred to her mind. She had been in a state of apparent anxiety for three months. A domestic difficulty was present and appeared adequate to cause her symptoms. She realized that this was the cause of her trouble, but the realization brought no relief. Hypnosis was attempted and completely failed. The cause of

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the failure, unknown then, has been revealed by her subsequent progress. She has gradually deteriorated and now shows marked signs of schizophrenia, and has had to be made a certified patient.

CASE 14.—Another illustration of the value of hypnosis for implanting desired suggestions :

Female, æt. 24. Married. Housewife. Voluntary patient.

This patient suffered from an acute anxiety state, consciously realized to be due to an induced fear of pregnancy. This had been systematically instilled in her since she was at school. When a domestic servant, her former mistress endeavoured to keep her services by painting to her in lurid colours the physical terrors of childbirth. She married a man earning a good salary. Her other sisters, not in such good circumstances, became jealous, and prophesied a mental breakdown for her when she should have a baby. She attended a sister's confinement, which further enhanced her fears and resulted in a psychological frigidity such that her husband remarked, "No wonder men go with other women".

She was agitated, tearful and depressed. Two months' psychotherapy without hypnosis enabled her to go out for week-ends, but not to leave the shelter of the hospital altogether. Hypnosis was induced. No new facts were elicited. Suggestions of confidence and disappearance of the fear of insanity were given and were successful. She left hospital in entire confidence, and has remained completely well ever since.

CASE 15.—The relief of insomnia by suggestion in hypnosis :

Male, æt. 43. Professional man. Voluntary patient.

Owing to domestic conflicts this man had become a drug addict. He had been taking veronal derivatives in large quantities both as sedatives and hypnotics. He was treated in hospital by abstinence. His attitude to his domestic trouble was adjusted. Nevertheless he persistently suffered from insomnia.

Hypnosis was tried with the specific intention of recapturing a lost sleep habit. He proved a good subject, and three sessions raised his sleep to seven hours a night, at which level he has continued since his discharge. He has successfully resumed his occupation, and states that he feels better than he has done for many years.

CASE 16.—Insusceptibility to hypnosis in another melancholic :

Female, æt. 34. Voluntary patient. Married. Home duties.

This was a case showing agitation, depression and feelings of loss of confidence, of four months' duration. The features made it difficult to decide whether she was suffering from melancholia or a severe anxiety state. There were several factors pointing to a psychogenic origin ; she had always been spoilt, and serious sexual difficulties had arisen between her and her husband. An operation was performed for the relief of dyspareunia following on the birth of two children, and it appeared as though her illness were a reaction to the knowledge that she now had no excuse for avoiding intercourse. As the most prominent feature was her lack of self-confidence, hypnosis was attempted

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in order to enhance the value of reassuring suggestions. Unfortunately she was entirely immune and could not be hypnotized to the slightest degree.

Subsequent events showed that she was in reality a case of agitated melancholia, from which she made a slow but complete recovery.

CASE 17.—Alcoholism; hypnosis used for exploration and attempts at readjustment:

Male, æt. 38. Voluntary patient. Labourer.

This patient had previously been in hospital, and had been regarded as an alcoholic and treated as such. He was compelled to leave, as he repeatedly returned drunk from town parole.

He was readmitted after four months, still alcoholic and having been drinking methylated spirits. Investigations in hypnosis revealed the fact that his alcoholism was associated with repressed fear. He was unable to go about the town without alcohol to give him "Dutch courage". He was liable to attacks of intense fear with all its distressing bodily symptoms when in the open street, and especially when in the presence of trams or buses.

Exploration showed that the origin of his fears lay in a series of accidents and explosions involving him and dating from his war service. He was wounded in the war, discharged from the army as unfit, and in 1917 was involved in a munitions explosion. Since then he had had an exceedingly narrow escape from being crushed at the docks between a ship and the quay. At the age of eight he had witnessed a child being killed by a tramcar in a busy street. The memory of all these occurrences except those connected with war service had been repressed.

Hypnosis recalled them to consciousness. He was given counter-suggestions of confidence, etc., and under their influence began to practise going about the streets again. His fears disappeared, and he regained control of himself as far as they were concerned.

Immediately after his discharge alcoholism returned acutely, although he no longer needed it to overcome his fears. He was repeatedly interviewed. Appeals to his moral sentiments, active sympathy and attempts to help him (he was now unemployed) resulted in a marked lessening of his craving for drink. He is at present at a Ministry of Labour training centre for the unemployed and is continuing to improve.

CASE 18.—Epilepsy; successful hypnosis without benefit to symptoms:

Female, æt. 18. Treated as an out-patient.

This girl suffered from epileptiform fits, with occasional incontinence and tongue-biting, of four years' duration. She did not always lose consciousness. There was great friction in her family, and the fits dated from a time when her mother had tricked her into going into domestic service in a convent.

It seemed a suitable case for investigation. Hypnosis was easily carried out on several occasions. She described fits in great detail and also the circumstances leading up to them. Counter-suggestions of sleep, steadiness and a more tolerant attitude to her mother were given, but the fits persisted obstinately. After trying these measures for two months without remission it was decided to regard it as a case of true epilepsy. This diagnosis was confirmed by the almost complete cessation of the fits on treatment by bromide

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and luminal. There was no family history of fits, and the girl (who remains well) has no features of the epileptic character.

CASE 19.—A remarkable case. Easily susceptible at first attempt and then totally resistant :

Male, æt. 49. Voluntary patient. Ship's captain.

This man was a clear case of anxiety neurosis. His duties demanded fine skill and judgment in attending to lightships. On one occasion in a storm he judged it expedient to leave a lightship unattended in safe anchorage. His employers disagreed and reduced his seniority. He was unable to face this blow to his self-regard, and developed severe symptoms. He was unable to take command in emergencies, was sleepless, and had headaches and pains in his limbs. There was a fine tremor of his fingers and face. These symptoms had lasted for four years.

The first attempt at hypnosis was completely successful and exploration revealed no evidence of repression. The interesting point about the case was that all further attempts failed completely. He was terrified at the prospect of resuming his former duties, and it seemed as if the failure could be correlated with an unconscious wish to remain ill and avoid having again to take charge of a ship.

Complete recovery commenced on receipt of a letter from his employers stating that he was to be invalided on pension from his work. He left hospital a fortnight later entirely well, but quite certain he could never take charge of a ship again, in case he had another error of judgment, and lost not only his job, but also his pension.

CASE 20.—Hysteria ; hypnosis used for exploration and as an aid to the reintegration of character by suggestion and persuasion :

Female, æt. 18. Voluntary patient. Domestic. Single.

This girl suffered from hysterical attacks, which took the form of staggering or falling down if spoken to sharply, or on unexpectedly meeting a smartly dressed man. It was later noticed that these attacks also followed any quarrelling amongst the patients in hospital.

The symptoms were so obviously psychogenic in type that hypnosis was forthwith resorted to with success. The source of the attacks was traced to an occurrence at the age of four, when she had suddenly come home and found her father beating her mother with a stick and forcing her to have intercourse against her will. The affect of fear was vividly aroused and persisted in the form of her present symptoms. It became clearly obvious that the sexual aspect of this event had never been repressed, because by the age of six she was seducing small boys of her own age from motives of curiosity and imitation.

Her symptoms were thus due to repression of the affect of fear, not of sex, with all due respect to the psycho-analysts. She was well aware of numerous sexual experiences which had no special emotional significance for her, and in our opinion, only a slight effect on the production of her symptoms.

She was now treated by persuasion. With her co-operation the genesis of her symptoms was explained, and she saw that there was now no further need for their persistence and gradually they disappeared. Successful attempts were made to integrate her character more fully and enable her better to withstand the stresses and strains of life.

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Discussion.

We have cited twenty very much condensed summaries from some forty cases with which we have attempted to deal by hypnosis. Necessarily these summaries have given only a sketchy outline to indicate the types of cases in which we found this technique of use. Failures have been included in accordance with the general principles governing the work, quoted in the opening paragraphs. In many cases it will be noticed that no specific diagnosis has been given. There are many reasons for this. Placing the emphasis on the psychogenic factor in early mental illness, and its corresponding therapeutic implications, immensely complicates the ordinary diagnostic criteria of descriptive psychiatry. Cases which have presented themselves in the first place as clear-cut psychoneuroses have later developed into or deteriorated into marked and inaccessible psychoses (e.g., Cases 3 and 13). The reverse process has also occurred (e.g., Cases 2 and 5).

Here we would point out what we find to be one of the values of hypnosis. Without exception, cases which presented as true psychoses could not be hypnotized. If susceptibility to hypnosis developed we were compelled to revise the diagnosis (e.g., Case 11). Similarly in Cases 3 and 13, when repeated attempts at hypnosis failed we were not surprised to find a bad prognosis. Speaking broadly, if hypnosis can be induced the prognosis is good, although the converse is by no means true (e.g., Cases 6 and 16). We have attempted hypnosis on many psychotics, invariably without success. We have failed on many psychoneurotics, but that has not meant that the diagnosis was wrong.

As regards results, we were able to induce hypnosis in 13 out of the 20 cases quoted. Of these 13, 11 recovered, and the recovery, to outsiders and relatives, is indubitable up to the present time; one is still under treatment (Case 8), and the other has improved on medical treatment (Case 18). Of the remaining 7 who were not amenable to hypnosis, 3 have recovered, 1 has died, 2 have definitely deteriorated and the last has been lost sight of. There can be no question that the use of hypnosis is a valuable agent in the treatment of these patients. It is becoming increasingly valuable to us with increased practice and better judgment in the selection of suitable material.

There is an opinion that hypnosis is attended by harmful effects. It is said that the patient becomes unduly dependent on the physician, and that his will-power becomes weakened so that he is unable to face life alone. We are quite unable to subscribe to this view, which fortunately is now rapidly becoming obsolete. True, an unscrupulous person might do a patient a great deal of harm by suggestion in hypnosis; and hypnotism as demonstrated on the stage does give the impression that the subject is totally under the dominance of the operator. With ordinary skill and care, however, and especially with that "inner goodwill for the patient" emphasized by Bleuler, there is

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no more danger in hypnosis than in the waking *rapport* which exists in all successful psychotherapy. One patient in our series (Case 9), incidentally not hypnotizable, for a short time incorporated in her delusions ideas of personal interference by us. Such a state of affairs is apt to occur in any mental hospital, as every psychiatrist well knows. For the rest we can categorically state that no patient has suffered any harm, or become unduly dependent on us. Indeed their discharge from hospital has been expedited. Our technique has been such that we have always avoided any attitude of omnipotence, and have encouraged patients to make their own decisions and face their perplexing situations themselves, merely equipping them with an unbiased attitude towards their troubles.

In our view the chief value of hypnosis lies in the fact that it enables one to take a short cut in the long and laborious process of mental exploration. The induction of hypnosis alone serves no therapeutic purpose, but the state is peculiarly suitable for the revival of repressed memories which have an emotional significance for the patient and a bearing on his illness (e.g., Cases 2, 5, 8, 10, 11, 17 and 20). Furthermore, the state of heightened suggestibility constituted by hypnosis enables therapeutic suggestions to be given which have a marked, if in some cases transitory effect in breaking the vicious circle of neurosis. In certain cases such therapeutic suggestions directed against specific symptoms seemed to be all that was required to turn the scale in favour of recovery (e.g., Cases 1, 14 and 15). We do not think, however, that such removal of symptoms is all that need be done. As full a mental exploration as possible is always beneficial; but with troublesome and worrying symptoms removed, such mental exploration is greatly facilitated, and our knowledge of the patient's individual difficulties becomes more complete. A point is reached when the patient consciously realizes how his own illness has arisen—a state of affairs which is the aim of all psychotherapy.

We are indebted to Dr. N. Moulson, our Medical Superintendent, whose interest and encouragement were ever readily given, for facilities to study and publish these cases.

References.—(1) McDougall, *Outline of Abnormal Psychology*, p. 465.—(2) *Idem*, *ibid.*, p. 480 —(3) Bleuler, *Text-book of Psychiatry* (1934), p. 220.

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Part II.—Reviews.

Anatomy, Physiology and Hygiene: A Textbook for Nurses. By A. MILLICENT ASHDOWN and E. BLEAZBY, D.N. London: J. M. Dent & Sons. Pp. 314. Price 5*s*.

Mental Nursing (Simplified). By O. P. NAPIER PEARN, M.R.C.P., L.R.C.P., D.P.M. London: Baillière, Tindall & Cox. Pp. 328. Price 5*s*.

Psychiatry for Nurses. By LOUIS MINSKI, M.D., M.R.C.P. London: John Bale, Sons & Danielsson. Pp. 79. Price 2*s*. 6*d*.

Hints to Probationer Nurses in Mental Hospitals. By R. EAGER, *O.B.E.*, M.D. London: H. K. Lewis & Co. Pp. 222. Price 8*s*. 6*d*.

Elementary Anatomy, Physiology, First Aid and Hygiene: A Preliminary Handbook for Nurses. Pretoria: Issued by the Department of the Interior. Pp. viii + 256.

Practical Psychology for Nurses and Other Workers in Mental Hospitals. By W. J. T. KIMBER, M.R.C.S., L.R.C.P., D.P.M. London: Humphrey Milford, Oxford University Press. Pp. 103. Price 3*s*. 6*d*.

The present moment, when the official Nursing Handbooks of the Association are about to be revised, seems an appropriate time to review a number of text-books and aids to nursing which have appeared recently.

The first on the list is designed to supplement the *Complete System of Nursing* written by its senior author, and probably the most widely-used work of its kind in this country. It is stated to cover the whole of the syllabus of the General Nursing Council in the subjects dealt with, and on perusing the mass of anatomical detail set out here, one is disposed to concede the claim without question. Whether the committing to memory of these facts is really a desirable preliminary in the training of a nurse is, of course, a matter that has been much debated, and the present book will not do much to allay doubts and anxieties on this score. It is difficult to believe that the selection of points for inclusion have been made with an eye entirely to their practical application; such details, for instance, as the histology of different varieties of epithelium, or an accurate description of the thoracic duct, might surely have been sacrificed without loss of efficiency in the nurse. On the whole, it is perhaps the predominance of anatomy over physiology that one would most like to see altered; only in a few places have the authors allowed themselves scope to put before the student the fundamental physiological principles involved, and here (as, for instance, in the section on respiration) they have done this admirably. It may be hoped that in subsequent editions they may multiply such explanations, and thus relieve the comparative aridity of the rest of their book.

One turns with some relief to the second edition of Dr. Pearn's companion

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to the R.M.P.A. Handbook. Here all is brightness and snappiness, and the author claims 150 reasons why the book should be more useful than the first edition, consisting of 25 subjects freshly introduced, 100 additional facts, and 25 minor alterations. A good example of one of these is in the chapter on hygiene, where an imaginary trip round a room fitted with every possible kind of ventilating appliance is described in a style that one can only call " breezy ". Constipation still reigns as the primitive first cause, capable of giving rise to mischief, yet not itself due to anything, and not apparently treatable. Dementia præcox is still given as the one condition connected with deficiency of the reproductive glands. It is to be hoped that this equating of the schizophrenic with the eunuch may not prove misleading, in more ways than one, to the nurse who may be disposed to take such a statement on trust. However, the book certainly serves its purpose well and deserves its popularity, and it sets a standard of attractiveness which the revisers of the Handbook will find it hard to attain.

Another small book, unpretentious in appearance and modest in price, is Dr. Minski's re-issue of a series of lectures originally written for the *Nursing Mirror*. This is addressed to the general nurse and gives a straightforward account of mental disorders, including as much psychology as is necessary for their understanding. As one would expect from the author, the psychiatric facts are well selected and the information up to date. Unfortunately he has not been well served by his printers, who have inserted as chapter headings what were meant to be headings of paragraphs only, with incongruous results. The description of the procedure for the admission of patients to institutional treatment does not quite fit that generally followed for rate-aided patients—a point of no great importance, however, since the reader is most likely to come in contact with private cases. If the book succeeds in tempting many general nurses to read some psychiatry and perhaps to pursue the subject further, its writing will have been more than worth while.

Dr. Eager's *Hints to Probationer Nurses* has been a well-known and much appreciated vade-mecum for many years. Its success has induced the author to expand it considerably in its new edition, so that it forms a text-book on mental disorders and their nursing treatment. It thus challenges comparison with the corresponding section of the " Handbook ", and other works covering the same ground. The description of the various forms of mental disease is not more exhaustive than that given in the Handbook, but it is on the whole clearer, and the salient points are well brought out. On the other hand the author has perhaps been rather too dogmatic in describing practices and conditions which do not universally apply, and in making use of the book to give publicity to views on what are not strictly nursing matters. It seems doubtful whether it is really advisable to include in a book of this type criticisms, however justified, of the ways of visiting committees, of the Board of Control or of the Mental Treatment Act.

A few surprising statements have crept in, which one hopes may be eliminated in a future edition. Such is the explanation of the term " dementia præcox " as referring originally to the belief that " these cases were all precocious at school ". One would also have thought that the pathology of cerebral tumours, of cerebral vascular disease or of encephalitis lethargica was at least as clear as that of general paralysis.

The malarial treatment of general paralysis is described in very great detail, but other recent special treatment methods are given little attention. Most surprising, in view of the author's known interest in the subject and the great

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reputation attained in its practice by his hospital, is the omission of any discussion of occupation therapy.

From South Africa comes what appears at first sight to be the *Nursing Handbook* itself, rebound in dark green. It is, however, the first instalment of its successor, produced by the medical staff of the mental hospitals of the Union in accordance with the syllabus of the independent State examination now held there. It is evident that the change in the examination has not meant any drastic revision of the syllabus or overloading of the curriculum. The teaching has been kept very simple ; in some places there has been further simplification compared with the R.M.P.A. Handbook, while elsewhere there is evidence of slight expansion. The presentation of the facts is somewhat clearer and more up to date, and more illustrations have been provided.

The nervous system is included in this preliminary handbook ; its description consists of the traditional enumeration of parts, followed by a simple account of its physiology, adequate as far as it goes, though hardly enough to aid the nurse in understanding the symptoms of neurological disease. In the section on hygiene it is noticeable that there is no reference to any special climatic or other conditions obtaining in South Africa.

Finally there is Dr. Kimber's slender volume entitled *Practical Psychology for Nurses*, in which the author has boldly attempted to compress a profitable psychopathology and a clinical psychiatry into not more than 100 pages. In this he has been, we think, remarkably successful, and the book seems to be the most satisfactory of those reviewed here. Dr. Kimber finds the Adlerian mechanisms the most useful to teach, but he refers to Freud too, and his exposition of both is convincing in spite of its brevity. The chapters on the psychological approach, or mental nursing proper, a subject often neglected even in books that purport to deal with mental nursing, are most valuable. One is left in this case with regret that the author should have set himself so rigid a space-limit, and with the hope that the inevitable expansion will not be too long delayed.

A. WALK.

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Part III.—Epitome of Current Literature.*

1. Anatomy and Physiology.

The Neuromuscular Junction. II. The Universal Antagonism between Calcium and Curarizing Agencies. (*Chinese Journ. Physiol., vol. x, pp.* 513–28, 1936 [*in English*].) *Feng, T. P.*

Ca antagonizes the curarizing action of such diverse agencies as the following: curare, eserine, veratrine, nicotine, atropine, ergotoxine, strychnine, pilocarpine and procaine, fatigue, acid, Sr, Mg, Ba and extreme temperatures. This universal antagonism between Ca and curarizing agencies is incompatible with Lapicque's chronological theory of curarization. The cause of curarization appears to be an electrical disturbance involving a loss of Ca from the neuromuscular junction.

E. D. Walter (Chem. Abstr.).

Studies on the Neuromuscular Junction. III. The Contracture in Eserinized Muscle is Produced by Nerve Stimulation. (*Chinese Journ. Physiol., vol. ii, pp.* 51–70, 1937.) *Feng, T. P., and Shen, S. C.*

A form of contracture in eserinized muscle is described, which has the following characteristics: it occurs only with tetanic stimulation of the motor nerve-fibres or their endings above a certain minimum frequency; it is localized to the innervated portion of the muscle, i.e., to the neighbourhood of the motor nerve-endings; it is abolished by atropine, novocaine and curare, i.e., by antagonists of acetylcholine. The effects of temperature and cations on the contracture were studied. It is shown that the most probable explanation available is that the contracture is due to acetylcholine liberated and accumulated at the motor nerve-endings and their vicinity, and so it is concluded that the eserine contracture furnishes strong evidence in favour of the theory of chemical mediation in the transmission of impulses from motor nerve to skeletal muscle.

L. A. Maynard (Chem. Abstr.).

The Liberation from a Stimulated Nerve of a Substance which Sensitizes the Muscle of the Leech to Acetylcholine. (*Boll. Soc. Ital. Biol. Sper., vol. ii, pp.* 741–2, 1936.) *Bergami, G., Cantoni, G., and Gualtierotti, T.*

An electrically stimulated nerve immersed in Ringer solution liberates a substance which sensitizes the muscles of the leech to acetylcholine.

Peter Masucci (Chem. Abstr.).

Effects on the Knee-jerk of Stimulation of the Central End of the Vagus, and of Various Changes in the Circulation and Respiration. (*Journ. Physiol., vol. lxxxviii, pp.* 459–75, 1937.) *Schweitzer, A., and Wright, Samson.*

The effects on the knee-jerk of stimulation of the central end of the vagus, lowering of blood-pressure, acute complete anæmia of the central nervous system, anoxia, alterations in the CO_2 pressure in the blood and the intravenous injection of various doses of histamine and cyanide were studied in the cat under chloralose anæsthesia.

E. D. Walter (Chem. Abstr.).

* A number of abstracts in this section are reproduced from *Chemical Abstracts* by kind permission of Prof. Crane, of Ohio University, to whom the Editors wish to express their thanks.

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2. Psychology and Psychopathology.

Tolstoy's Kreutzer Sonata and Unconscious Homosexuality. (*Psycho-Anal. Rev., vol. xxiv, p.* 18, *Jan.*, 1937.) *Velikovsky, P.*

The jealous murderer Pozdnaishef is presented as a homosexual who did not know his own nature. His Don Juanism prior to marriage; his unfounded jealousy towards his wife for ten years; the final situation, a creation of his own making, between his wife and the musician, the man he himself unconsciously desires; the musician's femininity with his hot clammy hands, red lips, slender build and largely-developed buttocks; lastly the sadistic nature of the murder, are all a cover for unconscious homosexuality. The author Tolstoy did not recognize this, nor did he understand the unconscious motives responsible for his creed that sexual love should not be practised. For Tolstoy sexual intercourse and even marriage were sins, and the highest ideal was complete abstinence and celibacy.

STANLEY M. COLEMAN.

Addenda to "The Medical Value of Psychoanalysis". (*Psycho-Anal. Quart., vol. v, p.* 548, *Oct.*, 1936.) *Alexander, F.*

This article and other new chapters are to appear in the new and revised edition of the author's *Medical Value of Psychoanalysis*. The present contribution is concerned mainly with organ disturbances and the theory of vector analysis.

Attempts have been made in the past to understand organ neuroses or diseases by an extension of the theory of conversion hysteria. It is psychologically unsound to interpret somatic phenomena that take place in the visceral organs as the direct expression of a specific psychological content, an emotionally charged idea or phantasy, in the way that Freud originally interpreted the significance of an hysterical paresis. In conversion hysteria the psychic tension finds direct expression in voluntarily-controlled organs—organs which are in permanent contact with the mental life in general and ideation in particular. In organ neuroses the emotional tension influences, via the vegetative nervous system, the function of vegetative organs. These organs have no direct relation to ideation and can only express general emotional trends. This is partly explained by the fact that the former psychoneurosis has its source in the phallic stage, when the child was capable of ideation; while the latter has its source in the pregenital stage, when the child's psychic life consists mainly of general emotional trends connected with the vegetative function. Both, however, are substitutes for voluntary action, representing the patient's disturbed emotional relation to his environment.

In the chain of causative factors determining an organ neurosis or psychogenic organic disease the following links can be distinguished: (i) chronic psychic stimulus (repressed tendency); (ii) excitation of sub-cortical centres through psychic stimulation; (iii) conduction of the excitation of the sub-cortical centres to different vegetative organs producing local changes in the vegetative function (organ-neurosis); (iv) morphological tissue changes under the influence of the chronic functional disturbances (psychogenic organic disease).

In studying the influence of psychological factors upon the vegetative functions the individual's general emotional trends and the dynamic direction of his impulses must be considered. Three major dynamic directors or "vectors" are isolated; these are the wish to incorporate, to eliminate and to retain. It is found that very different psychological impulses may lead to the disturbance of the same organ function, provided that they possess the same vector quality. It is believed that in the concept of psychological and biological vectors, the common denominator of psychological and biological processes has been found.

STANLEY M. COLEMAN.

Psychoanalysis and Conditioned Reflexes. (*Psycho-Anal. Rev., vol. xxiv, p.* 1, *Jan.*, 1937.) *Schilder, P.*

The data of Pavlov's experimentations are valuable and interesting, but his physiology is pseudo-physiology—popular mosaic psychology expressed in

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physiological terms. The Pavlovian use of such physiological terms as reflex, irradiation and inhibition to describe phenomena at a different biological level is critically examined. The Pavlovian reflex, for instance, is far from being a simple mechanical thing. It is a complicated total reaction in which the personality of the animal, as Pavlov himself has stated, plays an important part. All these reactions may be easily found in human beings too. It is a matter of taste to state that animal and human activity consists merely of reflexes, but the term "reflex" loses then every distinction, and is merely used to indicate that one is not interested in the subjective side of the animal's reactions but merely in the objective expression.

Either we have to give up the conception of the personality as a whole and of wholes and configuration in general, or we have to give up the Pavlovian idea which considers the cortical activity as a mosaic of excitation and inhibition. Animal experimentation can be sure of its results, and does not need any corroboration from the psychological side; but when animal experimentation invades the field of higher nervous activity and behaviour of animals, it should not forget that its result cannot be valid when contradicting acknowledged results of psychological research, and that psychological insight does not impair the results of objective investigation, but helps to their deeper understanding.

STANLEY M. COLEMAN.

Psychological Factors in Urological Disease. (*Psycho-Anal. Quart., vol. v, p.* 488, *Oct.*, 1936.) *Menninger, K.*

A small series of urological cases is presented, and an attempt made to investigate the contribution of emotional factors to pathological tissue alterations in the genital apparatus, particularly the prostate. All cases cited demonstrate the phenomena of a man whose emotional conflicts could tolerate neither a normal sexual life nor abstinence. His attempts at sexuality had masked destructive impulses, which were then reflected back upon him, first in the form of impotence (i.e., functional inhibition), and then, as if by replacement, with a more focal and autonomous self-destruction in the form of urethral and prostatic infection. From the few cases studied it appears that either staphylococci or gonococci may be utilized in the production of the lesions.

The hypothesis is that there is a self-destructive impulse in every individual which, hindered or fortified by other elements in the personality, achieves its purpose to varying degrees and in various ways. Its conflict with the life instinct results in compromises which may be regarded as partial suicides or focal self-destruction. These focal suicides may be carried out in a conscious deliberate way, as in self-mutilation, or in ways which seem accidental or extrinsically necessary, as in certain accidents and operations. These self-destructive tendencies, lying as they do deep within the fabric of the instinctual life, may also express themselves without the assistance of the voluntary nervous system and the striated musculature, and are to be seen as well in the death or injury of organs not directly connected with consciousness.

STANLEY M. COLEMAN.

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3. Psychiatry.

Genealogical Studies in General Paralysis [*Studi genealogici in famiglie di paralitici progressivi*]. (*Neopsichiat., vol. ii, p.* 507, *Sept.–Oct.*, 1936.) *Selzer, H.*

The family histories of 64 patients said to be suffering from general paralysis are considered and tabulated. The great majority show the presence of psychoses of various kinds in other members of the family.

H. W. EDDISON.

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Psychopathic Heredity, with Special Reference to Manic-depressive Psychosis [*Sull'eredita psichopatica . . .*]. (*Rassegna di Studi Psichiatrici, vol. xxvi, p. 3, Jan.–Feb.*, 1937.) *Ormea, A.*

This paper shows the results of a wide study of the hereditary transmission of psychoses, with special reference to manic-depressive states, which are regarded as being usually a recessive character. The author shows many reasons why preventive sterilization will fail to eliminate manic-depressive states.

Thirty-five genealogical tables are presented. H. W. EDDISON.

Manic-depressive Symptoms in Identical Twins [*Manifestazioni discordanti di psichosi maniaco-depressiva in gemelli identiche*]. (*Riv. Sper. di Freniat., vol. lx, p. 521, Dec.*, 1936.) *Selzer, H.*

Two cases of manic-depressive psychosis in twin sisters, aged 33, are described. One suffered from exclusively manic, and the other from exclusively depressive symptoms. H. W. EDDISON.

The Delusion of Intermetamorphosis [*Le délire d'intermétamorphose*]. (*Ann. Méd. Psych., vol. xv, p. 19, Jan.*, 1937.) *Daumezon, G.*

Report on two female chronic paraphrenics, aged 52 and 73, demonstrating the illusion of intermetamorphosis. The variations of Capgras's syndrome are considered to depend upon the degree of dissociation. In the illusion of negative doubles this is least advanced. While in Courbon and Tasques' case, where metamorphosis is complete, there is an extreme degree of dissociation. The two cases here presented take a middle place in the series, and correspond most closely to Courbon's Frégoli illusion. STANLEY M. COLEMAN.

Psycho-anæmic Syndromes [*Les syndromes psycho-anæmiques*]. (*Ann. Med. Psych., vol. xv, p. 177, Feb.*, 1937.) *de Morsier, G.*

During the past ten years the author has studied 17 cases presenting neuro-anæmic syndromes. Sixteen of these were women between the ages of 40 and 60; only one was a man. The cases were classified as follows: paræsthesia without other symptoms (3); paræsthesia, subacute combined degeneration, no mental symptoms (4); paræsthesia and mild depression (2); paræsthesia, subacute combined degeneration, Korsakov's syndrome (3); optic neuritis and visual disturbance (2); chronic psychoses (3).

This paper is mainly concerned with the psychotic cases. In one of these the anæmia first showed itself at the age of 48. Two years later the patient showed evidence of medullary involvement associated with an hallucinatory episode. At the end of 4 years, the blood-picture, neurological and cerebral signs having markedly improved, she had a further hallucinatory psychosis and died a few months later.

The second woman first showed signs of pernicious anæmia at the age of 39 with spontaneous recovery in 2 months. There was a further anæmia 6 months later with recovery after 6 months. Two years later she had a mental illness lasting 9 months; at that time the blood-picture was normal. Having remained well for 8 years she again showed evidence of anæmia with mild psychotic symptoms. Two years after, the blood-picture was normal, but the psychotic state became much more acute and she died during the next year. Her psychosis is described as a periodic form of schizophrenia. The most striking feature of this second case is that the psychotic state did not coincide with the anæmia, and that improvement in the latter was associated with more acute mental symptoms. The third case is to be the subject of a subsequent paper.

The pathogenesis of pernicious anæmia and the neuro- and psycho-anæmic syndromes are still unknown. The heredity factor, and the achlorhydric, neurological and vitamin theories are critically reviewed. It is noted that neuro-pellagric and neuro-anæmic syndromes have such a close clinical resemblance as to suggest a common origin. STANLEY M. COLEMAN.

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A Contribution to the Study of the Chronic Forms of Manic-depressive Psychosis [Contribution a l'étude des états chroniques de la psychose maniaque-dépressive]. (*Ann. Méd. Psych., vol. xv, p. 1, Jan.*, 1937.) *Halberstadt, G.*

Recent opinion upon the chronic forms of manic-depressive psychosis is critically reviewed. Chronicity means that the access is of unusual duration and should not be regarded as an indication of incurability. Some cases are chronic from the beginning; this is especially likely when the first attack occurs after the age of forty. It is generally found that the attacks gradually increase in duration, while the free interval diminishes and becomes less complete. Finally access and interval may become indistinguishable. In a case of some duration a bad prognosis is indicated by the absence of a perfectly normal interval, by fixity and diminished intensity of the clinical picture and by the presence of dissociation, stereotypy and automatism.

Terminal dementia from senile changes and especially from arterio-sclerosis may supervene and this also makes for a bad prognosis. A morbid heredity, especially schizophrenic, and an atypical physical constitution are other factors considered to make the outlook worse. STANLEY M. COLEMAN.

Observations on Heredity in Neurosis. (*Proc. Roy. Soc. Med., vol. xxx, p. 895, May*, 1937.) *McInnes, R. G.*

The writer gives us the result of his experience at the Jordanburn Clinic. His material consisted of 50 cases of anxiety neurosis, 30 cases of hysteria and 75 controls.

He finds that there are many cases of neurosis arising out of psychic conflict in which the patients appear to have no constitutional predisposition. In these cases the conflict is generally intense and activated by powerful exogenous stimuli. Recent cases of this type offer the best hope of recovery with psychotherapeutic help. At the other end of the scale are cases in which the patients have shown a consistent tendency to break down under ordinary or even trifling strains. In these cases the writer thinks it fair to assume the existence of a constitutional neurotic tendency.

He considers that his material furnishes confirmatory evidence of a general finding that neurotic patients are more heavily loaded than the general population, and in particular it suggests that in anxiety neurosis there may be a specific hereditary factor or group of factors which operate in some cases in producing emotional disorder. G. W. T. H. FLEMING.

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4. Neurology.

Trophic Disturbances in Hemiplegia. (*Z. Klin. Med., vol. cxxx, pp. 768–73*, 1936.) *Marinescu, G., Bruch, H., and Vasilescu, N.*

The endothelium of the capillaries is injured, and serum albumin and electrolytes (Na, K, Ca) pass into the tissue spaces, giving rise to œdema, hydrarthosis, urticaria, etc., and hindering diffusion of O to the tissue-cells for their respiration. On the hemiplegic side the serum albumin frequently is decreased.

JOSEPH S. HEPBURN (Chem. Abstr.).

Ascaris Infestation and Infantile Paralysis [Ascaridiosi e paralisi infantile]. (*Il Cervello, vol. xvi, p. 161, May 15*, 1937.) *Martini, F.*

Attention is drawn to the frequent coincidence of acute anterior poliomyelitis and ascaris infestation, an association stated to be so common that the author suggests that it is more than accidental. H. W. EDDISON.

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Tuberculoma in the Corpus Callosum Associated with One in the Basal Ganglia [*Tuberculoma del corpo calloso associato a tuberculoma dei gangli della base*]. (*Riv. di Patol. Nerv. e Ment., vol. xlix, p.* 115, *Jan.–Feb.*, 1937.) *Trabattoni, C.*

The author describes a case of two tuberculomata occurring in the same brain, one in the corpus callosum and the second involving the corpus striatum, thalamus, internal capsule and neighbouring structures. He discusses the evidence to show that the second was due to embolic spread from the original deposit in the corpus callosum. H. W. EDDISON.

Narcolepsy. (*Journ. Kansas Med. Soc., vol. xxxviii, pp.* 54–7, 1937.) *Speirs, G. O., and Speirs, R. E.*

The response of narcoleptics to ephedrine sulphate and to thyroid varies with individual patients. Of the four cases reported, two were responsive to ephedrine $\frac{3}{8}$ gr. daily, and the other two were aided more effectively by 1 gr. of thyroid daily. Ephedrine generally has a less lasting and uniform effect than α-methylphenethylamine (benzedrine). MARION HORN (Chem. Abstr.).

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5. Pharmacology and Treatment.

Phenobarbital Poisoning. (*Arch. Pediat., vol. liii, pp.* 670–5, 1936.) *Taylor, Herman W., and Schram, Maxwell.*

A boy, aged 10·5 years, received a total dose of 61 gr. of phenobarbital in 26 days, developed morbilliform dermatitis, hæmorrhagic, bullous and membranous stomatitis, pharyngitis, conjunctivitis, bullæ containing yellow fluid over the ear-lobes, and a blood-platelet count of 20,000, which returned to 300,000 in 14 days. Complete recovery occurred in 38 days. The cardiac condition (rheumatic endocarditis) of the patient became improved rather than worse. The treatment was essentially symptomatic. JOSEPH S. HEPBURN (Chem. Abstr.).

The Action of Narcotics on the Autonomic Nervous System. (*Upsala Lakarenforen Forh., vol. xlii, pp.* 209–43, 1936.) *Moraeus, B.*

Bromural, adalin, ethyl urethane and ethyl chloride had no effect on the autonomic nervous system. In studies on the frog heart and the rabbit intestine, dormiol, paraldehyde and chloralose have a strong affinity for the parasympathetic end-mechanism. On the frog heart threshold concentration of chloralose was 100 parts per million, paraldehyde 25c, and dormiol 1,250 parts per million. On the rabbit intestine the threshold concentrations were 375 to 500, 25 and 3 parts per million respectively. J. C. MUNCH (Chem. Abstr.).

Barbiturates. XX: The Alleged Anticomplementary and Anticoagulant Action of Barbiturates. (*Journ. Immunol., vol. xxxii, pp.* 35–41, 1937.) *Mollari, Mario, Randall, W. A., and Kappanyi, Theodore.*

The inhibition of complement hæmolysis and the prevention of coagulation of citrated plasma and of whole blood is effected by large but not by small amounts of Na barbital and Na phenobarbital, nor by large amounts of barbital and phenobarbital, nor by previous injections of large amounts of Na barbital into the animal. Na_2CO_3 in amounts sufficient to reach the H-ion concentration existing in the mixture after the addition of complement hæmolysis, and prevention of the coagulation of the whole blood or citrated plasma, show that the alkalinity of the medium is responsible. A. R. BEEBE (Chem. Abstr.).

Pathological Changes following Veronal and Medinal Poisoning in Rabbits. (*Trans. Soc. Path. Japon., vol. xxiii, pp.* 487–92, 1933; *Ber. Ges. Physiol. Exptl. Pharmakol., vol. lxxxii, pp.* 184–5, 1934.) *Nakamura, T.*

Acute poisoning produced by subcutaneous injections of 400 to 700 mgrm. of medinal per kgrm. failed to produce detectable pathological changes in the viscera

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of rabbits. Following daily administrations of 200 to 300 mgrm. per kgrm. chronic poisoning developed, associated with congestion, fatty degeneration and central necrosis of liver, fatty degeneration of the heart-muscle and pneumonic areas in the lungs. Slight but definite regressive degeneration was noted in the cerebrum and hind-brain. Oral administration of 1,000 mgrm. of veronal per kgrm. produced similar changes in the central nervous system.

JAMES C. MUNCH (Chem. Abstr.).

The Mechanism of Convulsions in Insulin Hypoglycæmia. Interrelationship of Blood Concentration, Cerebro-spinal Pressure and Convulsions. (*Amer. Journ. Physiol., vol. cxviii, pp.* 174–83, 1937.) *Drabkin, David L., and Ravdin, I. S.*

The events leading to convulsions in hyperinsulinism are severe hypoglycæmia → anhydræmia → rise in cerebro-spinal fluid pressure to a critical level → convulsions. Administration of Na arabinate, the practice of dehydration and excision of the stellate ganglia interfered with this cycle. E. D. WALTER (Chem. Abstr.).

The Effects of Tetanus Toxin on the Permeability of the Nerve-Centres. (*Boll. Soc. Ital. Biol. Sper., vol. ii, pp.* 768–9, 1936.) *Tsanto, A. D.*

Tetanus toxin, brought into direct contact with the nerve-centres (isolated cerebro-spinal axes of *B. vulgaris*), produced an increase in the diffusion of K. Compared with other exciting agents previously tested (strychnine, electrical stimulation), the increase in permeability was much more marked. This would seem to indicate a more pronounced effect of the tetanus toxin on the excitability of the control nerve-elements. PETER MASUCCI (Chem. Abstr.).

The Action of Adrenaline on the Knee-Jerk. (*Journ. Physiol., vol. lxxxviii, pp.* 476–91, 1937.) *Schweitzer, A., and Wright, Samson.*

Intravenous injection of 0·2–0·4 mgrm. of adrenaline usually produces in the cat a decrease or abolition of the knee-jerk accompanied by a decrease in muscle tone. Recovery sets in gradually and is complete in 10–90 minutes.

E. D. WALTER (Chem. Abstr.).

Parasympathetic Stimulating and Paralysing Actions of Acetylcholine-like Substances. (*Festschrift Emil C. Barell, pp.* 284–95, 1936.) *Blankart, A.*

In contrast to the parasympathetic stimulation of acetylcholine (I) and its lower homologues which give rise to increased secretion by the salivary, lachrymal and pancreatic glands, slowing of the pulse, reduction of the blood-pressure, increased peristalsis and miosis, an inverse atropine-like paralysis is brought about by higher homologues of I, such as $Ph(CH_2 : CHBrCH_2)CHCO_2CH_2CMe_2CH_2N(Et_2(Me)OH$, $Ph(CH_2OH)CHCO_2CH_2CMe_2CH_2N(Me)OH$, (II) and $Ph_2C(OH)CO_2CH_2CMe_2CH_2N(Et_2)MeOH$. The circumstances which bring about this reversal of action have been studied by the pharmacological investigation of the action of a series of acyl derivatives of choline, γ-homocholine and dimethylhomocholine on miosis, peristalsis, blood-pressure and salivary secretion. The dialkylaminoalkyl esters used in the preparation of the choline esters were obtained from the acid chloride and the corresponding alkanolamine. The α-hydroxyisovaleric acid and tropic acid esters were, however, prepared by heating $Me_2N.CH_2CMe_2CH_2Cl$ with the K salt of the acid in benzene. The $PhCH_2CO_2H$ esters were formed by re-esterification from the Me ester and the amino alc. The quaternary ammonium salts were formed by the action of MeI on the ester to give non-hygroscopic salts. Cholines in which the β-OH group is esterified are parasympathetic stimulants whose activity decreases with increasingly large esterifying groups. The conversion of the ethanol to the propanol chain so that the OH group is in the γ-position further lowers the stimulating action. This is still further decreased by the introduction of two Me groups in the γ-position further in the propanol chain, and the increase in molar weight of the acyl group is accompanied by change from stimulating to paralysing action. The introduction

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of an OH group into the acyl substituent group causes a decrease in activity, but is not responsible for the shift in action from stimulating to paralysing. The series shows that the transition from the stimulating action of I to the paralysing action of II is a gradual one, and that the intermediate compounds may cause a stimulation on some organs and a paralysing action on others. In attempts to connect the physical and physiological properties of this series of compounds, it has been shown that although the surface tension activity causes variations in effectiveness, it is not the sole determining cause. Solutions of 0·002 grm. mol. of the above compounds in 100 c.c. were tested for content by saponifying 40 c.c. with a known amount of 0·1 N. NaOH and back-titration of the alkali. The remaining 60 c.c. of solution was vigorously shaken for exactly 10 minutes with 2·0 grm. of defatted casein and filtered rapidly. The non-absorbed content of 40 c.c. of filtrate was estimated by saponification and titration and the absorbed material was estimated by difference. All compounds of which more than 3% was absorbed by casein paralysed the parasympathetic nerve-endings. The esterification of choline transforms it into a lipoid-soluble compound with stimulating properties, which are dependent on the rate of absorption on and elution from the tissues. This rate is varied by the size of the esterifying group. Highly lipoid-soluble and H_2O-insoluble substances with active combining groups are retained by the nerve end plate and, by blocking the nerve and hindering its function, cause paralysis. The physical properties of a drug not only decide its effective intensity but also decisively control the character of its effect. C. R. ADDINALL (Chem. Abstr.).

The Hypertensive and Hyperglycæmic Action of Hypertensive Cerebro-spinal Fluid Injected into Dogs after the Suppression of the Pressor-receptor Nerves. (*Boll. Soc. Ital. Biol. Sper., vol. ii, pp.* 704–7, 1936.) *Deleonardi, Stenio.*

By means of lumbar puncture, about 15 c.c. of fluid was obtained from six ordinary hypertensive subjects. The fluid was immediately injected intravenously into denervated dogs. In all cases there was a marked increase in arterial pressure. There was a marked hyperglycæmic action in three out of five cases. However, there seemed to be no parallelism between hyperglycæmic and hypertensive action with regard to the intensity of the reaction in the individual cases.

PETER MASUCCI (Chem. Abstr.).

Analysis of the Central Narcotic Nerve in Animal Experimentation. (*Festschrift Emil C. Barell, pp.* 394–406, 1936.) *Fromherz, K.*

The action of narcotics on the various functions of the nervous system as a whole and in part is discussed, and methods of animal experimentation and the difficulties involved are recounted. It is concluded that the final decision as to the suitability of a medicament can only be given by clinical trial. In the entire vertebrate series many effective qualities of drugs, such as those on the autonomic nervous system, are similar and for preparations, particularly those with some outstanding action, animal experimentation can afford excellent indications. Only for narcotics that have a general effect on the entire central organs can doses be transferred weight for weight from animals to human subjects. The indications are less trustworthy when the preparation acts specifically in some definite sense. Although drugs which have been used successfully in experiments on animals may fail in clinical applications and others may have therapeutic values which proved unfavourable on animals, the value of tests on animals is not to be understated, particularly if due caution is exercised in transferring the results to human therapy.

C. R. ADDINALL (Chem. Abstr.).

The Action of Dichlorodifluoromethane on the Nervous System of the Cat. (*Journ. Pharmacol., vol. lix, pp.* 176–81, 1937.) *Brenner, Charles.*

CCl_2F_2 is a generalized central nervous system depressant of low toxicity. Tremors following its inhalation are due not to convulsant poisoning but to " release " phenomena resulting from action on regulatory motor centres.

T. H. RIDERS (Chem. Abstr.).

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Use of Benzedrine Sulphate in Catatonic Stupors: Case Reports. (*Med. Bull. Veterans' Admin., vol. xiii, pp.* 224–7, 1937.) *Carlisle, Chester L., and Hecker, Charles H.*

Benzedrine (3-phenylisopropylamine) sulphate was administered to five patients and ephedrine sulphate to two for thirty-five days without therapeutic effect, contrary to the results obtained by Doyle and Daniels with ephedrine, and by Prinzmetal and Bloomberg with benzedrine.

O. HARTLEY (Chem. Abstr.).

Trypanocidal Activity and Arsenic Content of the Cerebro-spinal Fluid after Administration of Arsenic Compounds. (*Journ. Pharmacol., vol. lix, pp.* 157–75, 1937.) *Hawking, F., Hennelly, J. T., and Quastel, J. H.*

The intravenous injection of 3 grm. tryparsamide produces trypanocidal activity in the cerebro-spinal fluid in 14 hours, reaching a maximum in 40 hours, and disappearing after approximately 80 hours. Total As content of the fluid does not parallel activity, reaching its maximum in 14 hours and dropping rapidly. Apparently from 1 to 25% of total As is in active trivalent form. Other As +++++ compounds produced approximately similar results, while As +++ compounds led to lower concentrations of As and little or no trypanocidal activity in cerebro-spinal fluid. T. H. RIDER (Chem. Abstr.).

The Effects of Narcotics on Tissue Oxidations. (*Biochem. Journ., vol. xxxi, p.* 565, *April,* 1937.) *Jowett, M., and Quastel, J. H.*

The writers investigated the effects of luminal, chloretone and evipan on the respiration of tissue slices. With brain slices they found that respiration is more sensitive to luminal in the presence of glucose, lactate or pyruvate than in the presence of other substrates examined. The inhibition of respiration by narcotics tends to be independent of time when the potassium ion concentration of the medium is sufficiently high, while at a low potassium ion concentration the inhibition increases with time. Calcium ion influences the inhibitions. The inhibition due to chloretone develops rapidly after the addition of the narcotic and has no temperature coefficient. The substrate concentration has not been found to influence inhibitions of respiration by narcotics. The variation with narcotic concentration of the inhibition of respiration by narcotics follows a sigmoid curve.

Narcotics also inhibit oxidations by slices of liver, kidney and diaphragm, both when no substrates are added and in the presence of some substrates such as pyruvate. The respiration without added substrates is less sensitive to narcotics than is the respiration of brain in the presence of glucose. Narcotics inhibit the oxidation of butyrate by liver and that of alanine by kidney. The following individual stages in oxidation appear to be inhibited by narcotics: (*a*) oxidation of lactate to pyruvate by brain, (*b*) the oxidative deamination of alanine by kidney tissue. Narcotic concentrations which produce narcosis in vivo are of the same order of magnitude as those which inhibit measurably the respiration of the cerebral cortex in vitro. G. W. T. H. FLEMING.

Some Sympathetic Factors in Thermotherapy. (*Journ. Lab. and Clin. Med., vol. xxii, p.* 796, *May,* 1937.) *Alagia, D. P., and Flannery, D. L.*

The writers base their conclusion that bacterial protein fever therapy results in a primary parasympathetic stimulation followed by a more marked and prolonged sympathetic stimulation on the work of Hoff, who laid down the law that parasympatheticotonic conditions incline to leucopenia, a lymphatic tendency and eosinophilia, while sympatheticotonic conditions incline to leucocytosis, a myeloid tendency with a left shift of the nuclear count and a lowering of the eosinophiles.

G. W. T. H. FLEMING.

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Sleep Induced by Sodium Amytal: An Abridged Method for Use in Mental Illness. (*Amer. Journ. Psychiat., vol. xciii, p. 57, July,* 1936.) *Broder, S. B.*

The writer used sodium amytal for the induction of sleep in 58 patients. Several were treated with amytal and barbital as controls, but neither of these drugs was as effective. Patients tend to react differently on certain days, and the days on which they react most favourably should be used for intensive psychotherapy. The writer considers that the good results come from psychotherapy in the twilight state. Paranoid schizophrenics are very refractory to treatment, whilst those with simple schizophrenia and melancholia have little chance of improvement.

The "best day" is usually the third or fourth If convulsions follow the sudden withdrawal of the drug they are best treated with epinephrine ℥vij and morphine gr. iv. These convulsions, which are a form of "shock reaction", are frequently beneficial, i.e., of the 6 patients who reacted with convulsions, 3 had improved only slightly prior to the convulsions, but showed definite signs of recovery immediately after the convulsions. G. W. T. H. Fleming.

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6. Pathology and Biochemistry.

The Permeability of the Nerve Centres. II. The Effects of Crotalus Venom on the Permeability of the Isolated Cerebro-spinal Axes of Bufo viridis. (*Boll. Soc. Ital. Biol. Sper., vol. ii, pp.* 764–5, 1936.) *Marco, R. de*

Diffusion of K from the preparation immersed in Ringer solution is increased by the addition of crotalus venom to the medium. The effect can be intensified by electrical stimulation.

III. Variations in the Permeability of the Isolated Cerebro-spinal Axes of Bufo viridis by the Local Action of Morphine. (*Ibid., pp.* 766–7.)

Low concentrations of morphine-HCl in the immersion fluid decreased the diffusion of K; higher concentrations increased it.

IV. The Permeability of the Cerebro-spinal Axes of Bufo viridis under the Action of Trachinus draco and of Scorpæna scropha venom. (*Ibid., pp.* 767–8.)

The presence of either venom in the oxygenated Ringer solution in which the preparations were immersed increased the rate of diffusion of K out of it. The rate was still further increased by simultaneous electrical stimulation.

Peter Masucci (Chem. Abstr.).

A Study of the Cholinesterase Activity in Nervous and Mental Disorders. (*Quart. Journ. Med., vol. vi, pp.* 1–3, 1937.) *Tod, Henry, and Jones, M. S.*

There was a significant rise in the cholinesterase activity of the blood-serum in the anxiety neuroses and a fall in catatonic stupors and epilepsy. These changes may be due to corresponding changes in metabolism and the amount of acetylcholine produced. John T. Myers (Chem. Abstr.).

Ascorbic Acid Content in the Brain of Glis glis During Hibernation. (*Ukrain. Biokhem. Zhur., vol. ix, pp.* 879–89, 1936.) *Fomin, S. V.*

The ascorbic acid content of the cerebral hemispheres and cerebellum falls below the normal. It is assumed that the decrease in the oxidative processes during hibernation is connected with this phenomenon.

E. E. Stefanowsky (Chem. Abstr.).

The Influence of Chemical and Hormonal Factors on the Higher Nervous Activity of Castrated Dogs. (*Ukrain. Biokhem. Zhur., vol. ix, pp.* 489–505, 1936.) *Arkhangelskii, V. M.*

After castration the activity of dog cerebral cortex does not return to normal, either when left to itself or when different factors are applied to it. This is at least correct for animals of the inhibitory type.

E. E. Stefanowsky (Chem. Abstr.).

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Effect of Age on the Phosphorus Compounds of the Brain. (*Ukrain. Biokhem. Zhur., vol. ix, pp.* 613–32, 1936.) *Epel'baum, S. E., Khaikina, B. I., and Skvirskaya, E. B.*

In rabbit brain, in the first 7 days after birth there are considerably more total phosphate and acid-soluble P compounds than in adults. With increasing age there are decreases in the fraction hydrolysed during 7 minutes in normal HCl at 100° of the fraction of difficultly hydrolysable compounds and of creatinephosphoric acid. At 12–23 days the first two of these fractions are greater than in adults. After the 30th day after birth the content of P compounds attains the level characteristic for the brain of adults.

E. E. STEFANOWSKY (Chem. Abstr.).

Unimolecular Films of Nerve Proteins. (*Journ. Phys. Chem., vol. xl, pp.* 989–96, 1936.) *Fourt, Lyman, and Schmitt, Francis O.*

Nerve protein fractions were prepared. The general features of the unimolecular films formed by them on various sub-solutions are described. A phenomenon of pressure equilibration following change of area of these films was observed. This equilibration is not associated with changes in phase boundary potentials and is distinct from irreversible collapse. F. URBAN (Chem. Abstr.).

The Influence of Lactic Acid on the Cerebral Respiration of Mammals During Avitaminosis B_1. (*Biochem. Zeitschr., vol. cclxxxix, pp.* 136–42, 1936.) *Galvao, P. E., and Pereira, J.*

Lactate causes no increase in the O_2 consumption of the motor region of the cerebrum of rats with typical manifestations of B_1 avitaminosis, but in the cord the lactate exerts its usual effect. On the other hand in rats on a vitamin B-free diet without any manifestations of avitaminosis the motor area of the cerebrum shows the usual behaviour towards lactate, whereas lactate has no effect on the respiratory activity of the cord. The disturbances in birds are localized in the cord, while in the rat there are also disturbances in the motor area of the cerebrum.

S. MORGULIS (Chem. Abstr.).

Relationship between Brain Lipides and Learning Ability of Albino Rats. (*Journ. Genetic Psychol., vol. xlix, pp.* 389–403, 1936.) *Alm, O. W., and Whitnah, C. H.*

The scores for learning ability of rats obtained by maze tests gave positive correlations with the unsaturated galactolipides and phospholipides of the brain. The latter perhaps show a more uniform relationship to learning ability than the former. Other relationships were studied and are discussed critically.

WALTER H. SEEGERS (Chem. Abstr.).

A Study of the Chemical Composition of Various Divisions of the Nervous System. III. The Peripheral Nerves of Various Divisions of the Cow. (*Ukrain. Biokhem. Zhur., vol. ix, pp.* 69–84, 1936.) *Palladin, A. V., Rashba, E. Ya., and Gel'man, R. M.*

The chemical compositions of the anterior and posterior roots of the spinal cord per unit of dry matter are essentially the same; per unit of wet matter cholesterol, lecithin and cephalin contents of the anterior roots are lower than that of the posterior. In comparison with other divisions of the nervous system the absolute cholesterol contents are much greater in both cases. The inverse is the case for lipoids, nitrogen, creatine, P and water contents in the peripheral nerve. The N and creatine contents of the sympathetic nerve are higher than those of other nerves. It is concluded that a high protein and a medium cholesterol content are characteristic for the non-medullated nerves. The ganglia of the posterior roots have approximately the same composition as all the others, with the exception of a higher cholesterol and unsaturated phosphatides. Phylogenetically the

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cholesterol and unsaturated phosphatides are higher in older than in younger divisions of the peripheral and of the central nervous systems; the reverse is true of water and protein contents. E. E. STEFANOWSKY (Chem. Abstr.).

The Blood Sugar in Convulsions of Infancy and Childhood. (*Arch. Dis. Child., vol. ii, pp.* 257–60, 1936.) *Maclean, Margaret B.*

The concentration of blood sugar is frequently increased during or immediately after convulsions in infants and children. It then decreases rapidly to a level below normal, remains low for a period of several hours to several days and then rises to the normal level. The administration of adrenaline during the period of hypoglycæmia is followed by a significant increase in blood sugar. Except in cases of true hyperinsulinism, the disturbance in carbohydrate metabolism appears to be the result rather than the cause of the convulsions.

E. R. MAIN (Chem. Abstr.).

Patho-histological Studies of the Brain. I. Deposition of Lime. (*Sei-i-kai Med. Journ., vol. lv, no.* 9, *pp.* 1851–81, 1936.) *Shimada, Sadaaki.*

Of 34 cases in which the deposition of lime (D) occurred in the brain, only 1 case had D in the wall of blood-vessels alone; 25 cases, D in brain tissue only, and 8 cases D in both blood-vessels and brain tissue. Age is not a factor in D in brain.

S. TASHIRO (Chem. Abstr.).

Existence of a Complex Substance in Nerve Centres which Liberates Acetylcholine when Heated. (*Compt. Rend. Soc. Biol., vol. cxxiii, p.* 667, 1936.) *Corteggiani, E., Gautrelet, J., Kaswin, A., and Mentzer, C.*

Guinea-pig brain was triturated with a very dilute solution of eserine. The mixture gave a reaction with leech muscle corresponding to 0·25 mgrm. acetylcholine per grm. brain. The acetylcholine concentration did not change on standing at room temperature, but increased 4-fold if the mixture was heated to 70° C. for a short time. The acetylcholine content of an alcoholic extract of the brain was not changed by heating. Similar results were obtained with dog and rat brains and snail nerve ganglions. L. E. GILSON (Chem. Abstr.).

The Vitamin C Content of Cerebro-spinal Fluid in Neurological Patients. (*Klin. Wochenschr., vol. xv, pp.* 1528–9, 1936.) *Heinrich, Adolph.*

The Harris modification of the Tillman method was used. The variation in vitamin C content was no greater than in normal people. The older the patient, the lower the ascorbic acid content. WILLIAM MENDELSOHN (Chem. Abstr.).

The Cerebral Regulation of Sugar Content of Blood in Childhood. (*Orvosi Hetilap., vol. lxxx, pp.* 1139–40, 1936.) *Roboz, Pal, and Rausnitz, Erzebet.*

Blood-sugar content of children of both sexes and of ages 7–14 were determined by the methods of Hagedorn and Jensen (*C.A.*, 14, 2352). The normal blood-sugar level did not change significantly on treatment with 1·5 cgrm. pyramidone per kgrm. of body-weight in normally grown children. Large changes were observed in the vegetative nervous systems of backward children. Deviations exceeding 10-15% show a deficiency in the blood-sugar-regulating centre.

S. S. DE FINALY (Chem. Abstr.).

Bromine Content of Cerebro-spinal Fluid in Nervous Diseases and Insanity. (*Orvosi Hetilap, vol. lxxx, pp.* 1114–16, 1936.) *Nagy, Mihaly, and Straub, Janos.*

Numerous determinations according to the method of Leipert and Watzlawek (*C.A.*, 6393) showed that the Br content of the cerebro-spinal fluid varied between wide limits. The results seem to disagree with the statements of Zondek and Bier (*C.A.*, 4856; 27, 2201). Br content of cerebro-spinal fluid was not found to bear any definite relation to mental disease.

S. S. DE FINALY (Chem. Abstr.).

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Water, Calcium and Potassium of the Grey and White Matter of the Brain in Experimental Tetany. (*Compt. Rend. Soc. Biol., vol. cxxiii, pp.* 831–3, 1936.) *Parhon, C. I., and Cahane, M.*

The Ca and K of the brains of both normal and thyro-parathyroidectomized dogs vary within such wide limits as to make the results rather uncertain. The averages for the limited number examined indicate that after the operation the Ca of the grey and white tissues and the K of the white tissues increases. Changes in water content are not significant. Analytical data are given.

L. E. GILSON (Chem. Abstr.).

Chronic Experimental Catatonia Produced by Intermediate Products of Metabolism: Indolethylamine. (*Proc. Acad. Sci. Amsterdam, vol. xxxix, pp.* 1151–3, 1936.) *Nieuwenhuyzen, F. J.*

Intravenous injection of 100–150 mgrm. of indolethylamine-HCl in 3-kgrm. cats caused salivation and narrowing of the pupils combined with negativism and catalepsy. J. R. NELLER (Chem. Abstr.).

Carbohydrate Metabolism in Nervous and Mental Disorders. II. A Comparison of the Hyperglycæmic Index and Choline Esterase Activity in Anxiety and Depressive States. (*Edinburgh Med. Journ., vol. xliv, pp.* 46–9, 1937.) *Tod, Henry, and Jones, Maxwell S.*

In a group of 60 depressive psychoses there was no apparent relation between the emotional state and the choline esterase activity. In anxiety states the choline esterase determination gives a more reliable index of emotional disturbance than does the hyperglycæmic index; the more acute the case the higher is the choline esterase. RACHEL BROWN (Chem. Abstr.).

The Carbohydrate Metabolism of Brain. III. The Origin of Lactic Acid. (*Journ. Biol. Chem., vol. cxvii, pp.* 217–25, 1937.) *Kerr, Stanley E., and Ghantus, Musa.*

The free fermentable sugar of mammalian brain disappears within 3 to 5 minutes during post-mortem autolysis and 80–85% of the glycogen is lost within 15 minutes. The amount of lactic acid formed during a 2-hour period of anaërobic incubation corresponds to the loss of glycogen and free sugar; during the first 3 minutes the lactic acid production corresponds to the combined fall of free sugar and glycogen, and after that to the glycogen loss alone. The lactic acid maximum in autolysed brain from hyperglycæmic dogs is approximately equal to the blood-sugar level at death, the precursors being the free sugar of brain (equal to $\frac{1}{2}$ or $\frac{2}{3}$ of the blood-sugar level) and about 100 mgrm. of glycogen. In hypoglycæmic insulinized dogs the maximum, although low, is three times the blood-sugar level and again represents the amount of free sugar and glycogen available. It is concluded that both glycogen and the free sugar of the brain are precursors of lactic acid.

A. P. LOTHROP (Chem. Abstr.).

The Importance of the Mid-brain in Water Metabolism. (*Deutsch. med. Wochenschr., vol. lxii, pp.* 1905–8, 1936.) *Pette, H.*

The ingestion of a litre of fluid was fatal to two patients with brain tumours, due to the disturbed regulation of H_2O metabolism.

ARTHUR GROLLAM (Chem. Abstr.).

The Concentration and Excretion of Alcohol of Blood and Cerebro-spinal Fluid of Normal, Pellagric, Chronic and Acute Alcoholic Men and of Experimentally Alcoholic Animals. (*Zeitschr. Ges. Exptl. Med., vol. xcix, pp.* 341–51, 1936.) *Tomescu, P., and Dimolescu, A.*

A dose of 0·5 c.c. of 50% EtOH per kgrm. given to a series of patients was followed by determination of EtOH in blood and cerebro-spinal fluid. Pellagrins

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and chronic alcoholics remove the alcohol more rapidly than normal subjects. This is ascribed to altered permeability of the membranes.

MILTON LEVY (Chem. Abstr.).

Calcium Content of Different Parts of the Brain of Normal and Thyreoparathyroidectomized Rats. (*Biochem. Zeitschr., vol. cclxxxviii, pp.* 393–401, 1936.) *Farago, Stefan.*

Different portions of the brain of four groups of rats (normal, thyreoparathyroidectomized, operated animals treated with thyroxine and animals treated with parathyroid extracts) were analysed for Ca. There is a very great difference in the Ca content of the large hemispheres (6 mgrm.%) and of the cerebellum (0·4 mgrm.%). In thyro-parathyroidectomized rats the Ca content of the brain and cerebellum is increased but is descreased in the cord. Thyroxine administration has no effect on the Ca content of the operated rats, but parathyroid extract not only restores the normal relationship in the Ca concentration of the different parts but raises the concentration over the normal value.

S. MORGULIS (Chem. Abstr.).

The Influence of the Follicular Hormone upon Creatine Metabolism in the Muscular Dystrophies [*Influenza dell' ormone folliculare in soggetti miodistrofici*]. (*Riv. Sper. di Freniat., vol. lxi, p.* 101, *March*, 1937.) *Paleari, A.*

When cases of progressive muscular atrophy are treated with follicular hormone, creatinine metabolism is increased, with reduction of creatinine in the urine. This change was not observed in the other myopathies. These conclusions were based upon the examination of only a few cases. H. W. EDDISON.

Brain Phosphatase. (*Biochem. Journ., vol. xxx, p.* 1089, *July*, 1936.) *Giri, K. V., and Datta, N. C.*

Brain phosphatase shows two optima, at pH 9·4–9·6 and at pH 5. The ratio between the activities of the two phosphatases is almost constant for brains of animals of the same species. The activity of acetone-treated brain extracts is about as high as that of liver of the same animal. The phosphatase can be purified by (*a*) isoelectric precipitation of the inert material at pH 4·8, (*b*) ordinary dialysis, (*c*) ultrafiltration through cellophane membrane, and (*d*) acetone precipitation. Magnesium ions activate the alkaline phosphatase only. The percentage activation is distinctly enhanced when the enzyme extract has been previously purified by dialysis. Sodium hexosediphosphate is more easily hydrolysed than sodium glycerophosphate by the two phosphatases. G. W. T. H. FLEMING.

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7. Criminology.

Statistical Survey of Endocrinopathies Among Young Delinquents. (*Endocrinology, vol. xxi, p.* 189, *March*, 1937.) *Taylor, N. M., and Schaeffer, R. L.*

The authors examined a group of 2,311 delinquent adolescents, consisting of 1,106 females and 1,205 males. The percentage of endocrinopathies was almost evenly distributed between the sexes with a total of 17%, which is 7% greater frequency than Engelbach's estimated glandular dysbalance in the general population. The greatest number of cases occurred in the pituitary group. Hypothyroidism accounted for the greatest percentage of female endocrinopathies. Gonadism comprised about 14% of the total glandular disturbances and included almost 3½ times as many males as females. If the group of probable endocrinopathies be added to the positively diagnosed cases the percentage rises to 24%.

There is a marked increase in delinquency during the difficult period of puberty, and a more acute numerical elevation and higher incidence among the endocrinopaths at the time of puberty. G. W. T. H. FLEMING.

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CONTENTS FOR MAY, 1937

PAGE

Original Articles—

An Inquiry into the Incidence of "Neuropathic" Conditions in the Relatives of Normal Persons: Report by the Mental Deficiency Committee of the Royal Medico-Psychological Association 247

Diffuse White Matter Gliosis in Mental Defectives; by *A. Meyer, M.D. Bonn., and L. C. Cook, M.B.Camb., D.P.M.* 258

Aspects of Temperament in Adolescent Male Offenders; by *H. T. P. Young, M.B., Ch.B.Ed.* 268

Endemic Bacillary Dysentery; by *J. J. B. Martin, M.B.* 289

Hypnosis in Mental Hospital Practice; by *C. L. Copeland, M.B., Ch.B., D.P.M., and E. Howard Kitching, M.D., M.R.C.S., D.P.M.* . . . 316

Reviews 330

Anatomy, Physiology and Hygiene: A Textbook for Nurses; by *A. Millicent Ashdown and E. Bleazby, D.N.*—Mental Nursing (Simplified); by *O. P. Napier Pearn, M.R.C.P., L.R.C.P., D.P.M.*—Psychiatry for Nurses; by *Louis Minski, M.D., M.R.C.P.*—Hints to Probationer Nurses in Mental Hospitals; by *R. Eager*, O.B.E., *M.D.*—Elementary Anatomy, Physiology, First Aid and Hygiene: A Preliminary Handbook for Nurses (published by the Department of the Interior, Union of South Africa).—Practical Psychology for Nurses and Other Workers in Mental Hospitals; by *W. J. T. Kimber, M.R.C.S., L.R.C.P., D.P.M.*

Epitome of Current Literature 333

1. Anatomy and Physiology.—2. Psychology and Psychopathology.—3. Psychiatry.—4. Neurology.—5. Pharmacology and Treatment.—6. Pathology and Biochemistry.—7. Criminology.

CONTENTS OF SUPPLEMENT

The Royal Medico-Psychological Association.—South-Eastern Division.—South-Western Division.—Northern and Midland Division.—Irish Division.—Notices by the Secretary of the Research and Clinical Committee.—Notices by the Honorary Librarian.—Notices by the Registrar.

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THE
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No. 345 [NEW SERIES No. 309.] JULY, 1937. VOL. LXXXIII

Part I.—Original Articles.

MENTAL OBSERVATION WARDS:
A DISCUSSION OF THEIR WORK AND ITS OBJECTS.

By E. U. H. PENTREATH, M.R.C.S., L.R.C.P., D.P.M.,
Deputy Medical Superintendent, Derby County Mental Hospital, Mickleover; and

E. CUNNINGHAM DAX, B.Sc.Lond., L.M.S.S.A., D.P.M.,
Assistant Medical Officer, Leavesden Hospital, Herts.

INTRODUCTION.

FOR many years it has been the policy of the London Authority to avoid the certification of patients directly from their own homes, by passing them through an observation ward before making a decision as to their disposal. The system originated partly from the fact that the London Mental Hospitals are some distance from town, and partly from the shortage of accommodation, it being necessary to secure the safe custody of the patients pending their admission. The small scattered wards have now been fused together to form larger and more efficient units, which ensure every case having the fullest consideration and investigation before deciding upon any method of disposal. The various points arising in this paper are illustrated by reference to the work of the largest of these units.

We feel that to the psychiatrist a description of the extraordinary variety of patients passing through an observation ward, the procedures involved in their sorting and distribution and the figures of their ultimate place of disposal might prove to be of interest. By incorporating these points in a discussion of two recent publications, we hope that the paper may have a wider scope and perhaps be of some practical use.

In the Maudsley Hospital Medical Superintendent's Report, 1932–35, it is shown how the development has taken place of " a co-ordinated system of dealing with mental and neurotic diseases of all kinds, and degrees, and at all stages ". In Section 1 we have attempted to outline the place of the

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observation ward in this scheme, and to describe briefly the management and planning, and the steps taken to avoid mixing cases that might adversely affect one another. Various other references to this report will be found in the different sections.

The Board of Control's Report for 1935 (Part I) contains several references to the observation wards in municipal hospitals and institutions. The investigations of these, it says, is disquieting on the whole ; in some, newly admitted patients are mixed with chronic cases and low-grade defectives, and the staff have not had the training necessary. Reference to this criticism, as mentioned above, is made in Section 1.

The latter report begins by stating that the overcrowding in mental hospitals is more serious than the net figures suggest ; and adds later—" Once it is clear that the patient requires treatment for mental illness, no time should be lost in transferring him to the mental hospital, which in general is the only place able to provide the specialized experience and the therapeutic resources necessary for successful treatment ". Both these points are raised in Section 2, which discusses the advisability of immediate transfer, the procedure in regard to each individual case, and the powers of the visiting Justice of the Peace.

A classification of the various types of disorders in the admissions is given in Section 3 as a matter of interest. The vast majority were genuine cases of mental illness, each clearly in need of treatment, but surely one is not to transfer them all to mental hospitals ? The above statement from the Report must, in spite of its definite wording, be somewhat broadly interpreted.

In Section 4, which relates to the disposal of cases, the figures are analysed. The points in the report with reference to the Mental Treatment Act, and the use of temporary treatment in particular, are considered.

Finally Section 5 is devoted to a short discussion of some conclusions and proposals which have arisen in the work.

In this paper it is suggested that well-organized observation wards, situated within easy reach of the most populated areas, can deal suitably with doubtful cases coming to general hospitals and local doctors. Also, that it would no longer be necessary for relieving officers, police and other officials to have to make a decision in suspected cases of mental disorder brought to their notice. Some modification of the system might have to be made in rural districts. Moreover, the observation wards can obviate the difficulty of deciding the line of action to be taken in the event of further treatment being necessary, and diminish the number of people unnecessarily certified.

In addition, we believe that if certain cases were adequately dealt with during the temporary powers of detention in a well-organized observation ward (at present limited, by a relieving officer's 3-day and a medical officer's 14-day order, to 17 days), they would have no need of mental hospital treatment. A further passage from the Board of Control's report runs : " Every improvement of the observation wards increases the temptation to undertake active

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treatment, a practice quite inconsistent with the main purpose of such wards, which is the diagnosis of doubtful cases." Whilst agreeing with this statement for most observation wards at present, we believe that in the future reasonably large units will provide an adequate classification of cases, and give the opportunity for time, skill, experience and patience to be used, to gain the co-operation of the patient and avoid his transfer to a mental hospital. Whatever the views expressed as to the advantages of the latter may be, there can be no doubt about the objection of patients and their relatives. Moreover, in transient psychoses, whether volitional or not, recovery can take place with efficient treatment in the observation ward, enabling the patient to return to his daily routine, perhaps with the aid of an out-patient clinic. In chosen cases we believe that time should also be given to regain volition, or insight and better judgment, so that the patient may then co-operate with the doctors and authorities.

The following description refers to an advantageously placed observation ward in the service of the London County Council, and includes an analysis of the types and their relative numbers among over 1,100 admissions in a little more than a year.

Section I: Description.

The mental block was under the administration of the Public Health Department of the London County Council, and the largest of such units. It consisted of 82 beds disposed in two similar male and female blocks, renovated in recent years with the object of taking over the work of several smaller wards in other institutions and hospitals. About 1,000 cases a year were thus dealt with. Adjoining, and under the same administrative control, was a large general hospital and a chronic sick institution.

The unit drew its patients from two public assistance areas. The one with a population of 436,893 supplied 447 cases to this ward in a year and 184 to others; 113 of the patients were certified. From the 296,147 inhabitants of the other area, 123 of the 482 admissions during the time were certified.

In each block new cases were admitted to a ward of ten beds for preliminary observation; adjacent to it were two shuttered side-rooms, two warmed and lighted padded rooms, lavatories and a bathroom with continuous bath. Thus this portion was self-contained, and so avoided the disturbance of patients in the remaining wards.

Padded rooms are often condemned, but they prove especially useful in this work for really violent and for restless senile patients; moreover they diminish the number of staff who would otherwise be required to keep the cases in bed, and save the extensive bruising which is bound to arise if this is done, besides avoiding an atmosphere of antagonism and unrest which is always bred by constant skirmishings.

Padded beds with high sides, as used in certain of the L.C.C. observation

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wards, covered in by netting if necessary, have much to recommend them, if used in addition to, rather than in place of padded rooms. They should be used in a single room to avoid disturbing and perhaps frightening other patients. They have several distinct advantages: they allow less freedom than does a padded room, and thus there is less chance of injury, observation is easier, struggling upon entry is no longer necessary, and the room may be used as an ordinary side-room when not required by the excitable or restless type of patient. On the other hand there is probably more difficulty in keeping the patient clean, and the excessive restriction may upset certain cases who settle in the ordinary padded room, perhaps without seclusion. It is to be noticed that this method of dealing with excitable cases does not constitute a form of mechanical restraint.

Another rather larger ward was reserved for quieter bed cases; there were, in addition, kitchens and offices on the ground floor. Above these was a further dormitory and a comparatively large recreation room with facilities for ping-pong, various other table games, a wireless set and a piano. It was decorated with attractive bright pictures, framed copies of pictorial railway posters, which lend themselves admirably for decoration of this kind. An airing garden provided daily exercise. The size of the wards, the mixed types of cases and the rapidly-changing patients render the usual forms of occupation therapy inapplicable. The nursing staff do most valuable work in encouraging light simple handwork, and various forms of useful employment, on the medical officer's advice; in most cases any but the very lightest forms of domestic duties are not advocated.

The medical officer in charge had had several years' experience in the Council's mental service, and was seconded to the unit; incidentally he received a fixed salary, and had no remunerative interest in the disposal of patients under any form of recommendation or certificate. He was aided by a second medical officer and a trained psychiatric social worker.

The majority of the nursing staff were certificated in mental work or had had mental hospital experience. A relatively large staff is necessary because of the different and constantly changing types of case. Their work is more difficult than that of the nurse in charge of most of the more permanent occupants of mental hospitals, as they have continually to adapt themselves to new patients. Considering this, at least a portion of their necessary training must be in the observation ward, and we expect that in the future a period of such service will considerably enhance a nurse's qualifications. As in mental hospital routine generally, the maintenance of a good tone conducive to the patients' self-respect is desirable in observation wards, and the attitude and manner taken up by the staff towards the patients individually and generally should be one of civility and sympathy.

This unit was singularly fortunate from the viewpoint of investigation and prognosis in having the facilities of specialized advice and treatment adjoining,

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in all branches of medicine and pathology, and the opportunity for interchange of cases with the acute and chronic sick wards.

The proximity of the Maudsley Hospital, with its special annexe at King's College Hospital, provided facilities for close mutual co-operation. Thus, cases were sent there as in-patients, others on discharge went to the out-patient clinic, and many were admitted from the latter department for transfer to mental hospitals.

The Medical Superintendent of the Maudsley Hospital visited as Consultant twice weekly, and gave advice on the difficult cases, patients with a hopeful future and those electing to go away for voluntary treatment. In addition he saw about 100 other cases each year sent from the general hospital for his opinion.

On the other hand, from the point of view of the mental hospital, we made an attempt—

(*a*) To decide the disposal of cases with due regard to their prognosis. Thus patients who were likely to recover in the course of a few days were kept in the ward.

(*b*) To use the legal processes of transfer in such a way that re-grading would be unnecessary.

(*c*) To transfer patients in as good a state of physical health as was possible.

With regard to this last point, the state of neglect in which a large number of cases arrive at the observation ward is unbelievable to those who do not admit them. This applies particularly to some senile patients who have been living alone. Their clothes have not been taken off for months, they are alive with vermin and filthy with excretions. Their mouths are dirty, they are half-starved, constipated, bronchitic, anæmic and toxic. It is a wonderful tribute to the nursing staff that there is such a magical change in the patient's condition so soon after admission.

Thus the observation ward, instead of being small, isolated and ineffective, has come to be, we believe, a useful part of the machinery for dealing with mental disorder.

Section II: The Management of Each Case from Admission to Discharge.

Most of the cases were admitted by one of the following procedures:

1. A relieving officer of the district supplied a legal detention order lasting for three clear days (Lunacy Act, Sect. 20) unless the patient was discharged within this time by the justice of the peace. In a series of 500 consecutive cases, 378 (or 75%) were thus admitted, including 33 cases of attempted or threatened suicide and 2 of homicide.

2. Police officers gave a similar order admitting 54 cases (or 11%). These

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were usually more violent, and amongst them were 12 suicidal attempts (4 by drowning), 12 who were found wandering at large, 8 cases of violence, 7 alcoholics, and 6 who were behaving in an odd manner.

3. An institutional medical officer's order in writing (Lunacy Act, Sect. 24 (1)), which would detain a patient for fourteen days. These entries came from the general hospital through the chronic sick wards, or from them directly. This order, moreover, was used to detain the patients in the observation ward when the Justice made " no order ".

4. By the order for pauper lunatics wandering at large (Lunacy Act, Sect. 15). There were only two such admissions.

5. A few entered of their own accord ; this is an interesting development, and shows an increasing confidence in the treatment, and an absence of that horror with which the public are supposed to regard the experience. If a person enters an observation ward in this way, he is under no obligation to give any period of notice before leaving, and is not recognized under the legal status of a " voluntary patient ". For this reason the system is obviously open to abuse, and judgment has to be exercised, otherwise it would become a simple method of obtaining a night's lodging. Actually this did not concern us, for the only patients thus admitted were from the administrative unit.

A Justice of the Peace (on a rota of duty) visited the ward three times weekly and saw all cases within three days of admission, and others of longer standing, submitted for legal certification. He either made an order for the patient's reception in a mental hospital after interviewing him and considering the medical officer's certificate, or alternatively he made no order and signed a book to that effect. He nearly always asked the medical officer's advice, and generally it was accepted. Evidence was supplemented by police or relieving officers, and the relatives, whose views as to certification and treatment were considered, after the position and the procedure advocated had been explained to them. An experienced justice would adequately stress the possibility of serious consequences arising if the advice were not taken, and he could be a great help with certain relatives, who sometimes presented greater problems than the patients themselves.

Certain types of case are best certified without delay, and 45 of our series of 1,156 were dealt with thus within the first three days of their admission. Sometimes it was difficult for such reasons as the following :

(1) Some cases had been certified before and knew the routine, consequently they were not prepared to give themselves away to the Justice of the Peace, at least on their first interview.

(2) Suicidal or homicidal patients might conceal their intentions.

(3) Paranoid patients might undergo temporary improvement on change of surroundings.

On the other hand, it was one of the first principles of disposal not to transfer a patient to a mental hospital if it could be avoided. Acute cases

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nearly always showed a rapid improvement. A very remarkable case illustrating this point was a post-influenzal hallucinosis who chased lions and picked strawberries from the walls of the padded room, but recovered two days later and did not relapse. (This was his first illness and there was no alcoholic or drug history.) Alcoholics needed some days' treatment, but were usually uncertifiable after a very short time.

If all patients of these last two types of case were transferred because of an acute transient psychosis or delirium, a large number of patients would be sent to mental hospitals unnecessarily. It will be seen in Section 4 that it was possible to discharge about a quarter of the total admissions, usually by the end of seventeen days, thereby avoiding an enormous burden on the already overcrowded mental hospitals.

Those cases best certified eventually, such as the first three examples, have often to be watched carefully for days, or weeks, before their true psychotic nature is revealed.

At his first interview with the patient the Justice usually made "no order", and a medical officer's fourteen-day order was completed, giving up to a total of seventeen days in which to diagnose, treat if necessary, and form a reasonable prognosis of the case.

At the time of the patient's admission to the observation ward a short history was taken from the relieving officer or police, and the relatives or friends. A reasonably thorough physical examination was made, notes taken of injuries, and as soon as possible, some details of his mental state. After bathing, the patient was put to bed in the admission ward for observation, and reports were made by the staff on his conduct, tendencies, delusions and hallucinations.

The medical officer's first duty was to decide the following questions in the light of the past history and present type of illness:

(1) Was it justifiable to certify the patient immediately?
(2) Was he an obvious case to go away as a voluntary patient?
(3) Would he make a suitable temporary patient?
(4) Was he in need of treatment at a hospital for senile cases?

If these questions could be definitely answered no time was lost in making the necessary arrangements, as suggested in the Board of Control's Report. The majority, however, gave no such clear indications, and needed further investigation and observation before these points could be decided.

In cases other than those certified within the first three days fuller mental and physical notes were made, and routine Wassermann reactions and any other pathological examinations thought necessary were done. They were treated for such conditions as insomnia, constipation, dehydration, oral sepsis and gross cardiac or pulmonary disease, special attention being paid to the confusional and delirious cases. Most cases improved to an extent whereby the underlying psychosis was revealed, allowing a fairer prognosis and classification to be made. The indiscriminate use of sedatives merely for the sake of keeping

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the patient quiet was regarded in much the same light as is giving morphia to one with an acute abdomen before a diagnosis is made or indication for operation is clear. The questions of bromides will be discussed later.

In some cases encouragement and suggestion were employed, co-operation gained, and with the help of the change of surroundings, rest and recreation, insight was obtained. By this means a large proportion would either go as voluntary patients to mental hospitals (if not discharged home as recovered), or if they improved sufficiently were put under out-patient care at the Maudsley before, or soon after, seventeen days.

As stated above, the wishes of the relatives were usually fully considered, to gain their co-operation, and to remove the fears which are often associated with mental hospitals. The friends and relatives like to think that there is nothing "mental" about the patient, but that the cause of the trouble is merely a "nervous breakdown" or "nerves", however bad he may be, and what the neighbours may say often counts for a good deal. In this connection it is to be noted that the London County Council have recently changed the names of their mental hospitals, omitting the "mental" portion. This is an improvement, but much public re-education will have to take place, in addition to changes in the system of dealing with mental disorder, before the hospitals are generally accepted.

The social worker investigated the history of many of the cases, often interviewing relatives or friends in their own homes, so that a better idea of the domestic conditions could be obtained. A shrewd but tactful and pleasant manner in the experienced social worker will enable her to detect unreliable or inadequate statements given by frightened or difficult informants. The worker trained in psychiatry will note schizophrenic, paranoid or other relevant symptoms given in the story of the patient's habits of life and the circumstances leading to admission. She will also obtain a family history, the details of the patient's early life, his work and his financial position, and set it out in a concise form. A copy is kept with the patient's notes, and a duplicate is sent to the hospital to which he goes. Such a history is essential in making a reasonable prognosis in a border-line case; it does not differ much from the work done at the mental hospitals, but has to be completed within a short time. The medical officer has not the time necessary to do such work as efficiently as can the social worker. She is also invaluable in making arrangements for patients going away on a voluntary basis to mental hospitals or to After-Care Association homes. Often she can place those who are in need of a home or hostel but not of mental hospital treatment, or even find suitable employment for some cases. She can keep in touch with patients who may relapse after discharge, and this in the observation ward work is a very important part of her duties. She can co-operate with social workers in other hospitals, obtain all sorts of odd information, such as is done by the Lady Almoner in general hospitals, and keep "follow-up" records.

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When the history of the patient and his examination were completed he was seen by the consultant psychiatrist, who gave a prognosis and advised upon disposal, or suggested further investigations and observation.

Young people in their first attack always received special consideration, and the social worker could help a great deal by following them up. The alternative methods of disposal, which will be discussed in more detail in Section 4, where the results are analysed, were briefly these :

1. To go home, or to an after-care or convalescent home, perhaps after a slightly extended period in the ward on a voluntary basis.

2. To go to a general hospital, an epileptic colony, special home, hostel or institution.

3. To go as a voluntary patient to the Maudsley or to a mental hospital.

4. To be recommended as a temporary patient.

5. To be certified (under the Lunacy or Mental Deficiency Acts).

6. To be sent to a hospital for senile cases. In the London County Council service there is a special hospital for the more docile type of senile patients.

Section III : The Type of Case Admitted.

Although some of the groups of a classification must overlap, the following brief scheme gives an idea of the types of cases admitted, and their relative numbers. The figures below were found for the 518 cases with which one of us dealt :

	Males.	Females.
1. *Mental deficiency*	10	2
2. *Organic nervous and mental disorders :*		
a. Senile changes	52	52
b. Cardio-vascular :		
1. Heart failure	6	3
2. Other conditions	7	2
c. Various gross central nervous system lesions	8	6
d. Epilepsy	12	4
e. Neuro-syphilis :		
1. Interstitial	2	1
2. G.P.I.	10	6
3. Tabes	1	1
f. Infections, endocrine and other physical disorders	14	9
g. Alcoholism	27	18
h. Bromide intoxications	4	6
i. Confusional states of doubtful origin	7	7

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	Males.	Females.
3. *Depressive states*	32	53
4. *Manic states*	11	21
5. *Schizophrenic states*	48	41
6. *Moral abnormalities*	3	3
7. *Psychoneuroses*	8	8
8. *Institutional cases*	12	1

Syphilis.—Of the 274 male cases 24 had a positive blood Wassermann reaction, but only 10 of these were general paralytics. Two had interstitial syphilis (one an amyotrophy) and another was a tabetic. Among the 224 females there were 13 with a positive blood Wassermann reaction. Six of these were general paralytics, one a tabetic and one a case of cerebro-spinal syphilis.

Three of the 16 general paralytics died before having treatment, 2 had previously been unsuccessfully treated with malaria, whilst 2 others refused treatment and were not certified. Of the 9 who went away for malaria therapy, 3 made a good recovery.

Alcohol.—Three males and 4 females were admitted in a state of intoxication secondary to a psychosis, and all went to mental hospitals.

These are not included in the above figures for the alcoholics. The total number formed nearly 10% of the admissions, and were interesting in being so different from the ordinary inebriated person that they were brought for observation. The relative forms of the disorders in the sexes were :

	Males.	Females.
Delirium tremens	4	1
Confused, hallucinated and paranoid	15	12
Alcoholic pseudo-paresis	2	—
Amnesic and demented	2	5

Three of the males were certified and 2 went away as voluntary patients. Five of the females were certified. In addition, 4 old men (3 over 75) were brought in after " celebrations " and later discharged.

A point to which much recent attention has been paid is " Red Lizzy " or " Red Biddy " drinking. We could find 2 males and 7 females who drank little else than cheap red wine ; in no case were there any eye signs, nor in fact was there anything typical about them other than this strong addiction. It seems that some confusion arises in the distinction between " Red Lizzy " on the one hand and cheap colonial red wine and Lisbon wine on the other, and that they are wrongly called by the same name. " Red Lizzy " was originally described as cheap red wine to which methylated spirits had been added by the drinker, though it has never been found to be sold in this way.

Bromide intoxication.—Routine blood bromide estimations were made on the last 240 admissions: 14 of these were higher than 150 mgrm. %. Each of

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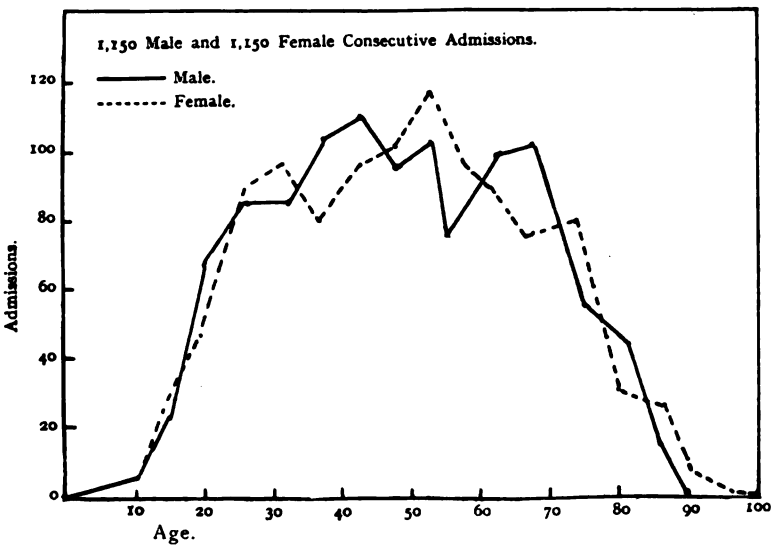
these patients was given oral saline, and those above 200 mgrm. % rectal salines in addition. It is surely a very suggestive, though not conclusive, fact that 4 of these 14 cases died within three weeks of admission! Their ages were 76, 67, 63 and 52, and their blood bromides 175, 160, 150 and 210 mgrm.% respectively. Within this period 81 other patients aged 60 and over were admitted and only 7 of these died. After a variable time in the ward 5 of the 14 cases recovered sufficiently to return home.

The statements made upon the increasing facility of intoxication with age appear to be well confirmed by these observations.



Other cases of note.—Among the gross central nervous system lesions there were cases of cerebral tumour, cerebral angioma, head injury, disseminate sclerosis, Huntington's chorea and Parkinsonianism.

The sixth group, among the organic disorders, contained patients with phthisis, post-operative disturbances, hyperthyroidism, myxœdema, Simmond's disease, acromegaly, jaundice, uræmia and diabetes.

Pathological liars with some transient hysteria (usually young women), and social problems such as police-court cases, the homeless and the friendless, found their way into the ward from time to time.

Institutional cases.—It will be seen that a separate group has been made for the cases coming in, for instance, from casual wards, institutions, and the streets. A few were obvious malingerers; others, people who needed some supervision, the types who went in and out of jobs, but were usually unemployed, and for whom one could do little to help. These were clearly unsuitable cases either for the mental hospital or for the observation wards and they were usually rapidly returned to the place whence they came.

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Age-incidence.—It may be of some interest to show the age-incidence of the admissions, which perhaps gives a truer distribution than the figures taken from those cases going to mental hospitals. The curve below is the result of plotting the mean ages against the admissions for each five years (*e.g.*, age 23–27 as 25, 28–32 as 30). To give it more value, we have added the 1,100 previous cases to our own (see diagram, p. 357).

Section IV: The Disposal of Cases.

The various alternative methods of disposal have already been mentioned; the following figures are quoted from 1,156 cases (638 of one writer and 518 of the other). The numbers were reduced by local circumstances, such as the temporary overcrowding of the hospitals for senile cases; these patients occupied for lengthy periods many beds which would otherwise have been used for younger psychotics. Cases from our own areas had at times to be sent to other observation wards, in consequence of this congestion. In the analysis below the writers (one of whom followed the other in charge of the ward) worked independently without having the present paper in view. The agreement of the figures is so remarkable that the percentages are quoted independently under columns 1 and 2.

	638 cases. I. (%.)	518 cases. II. (%.)	Combined number.	1,156 cases. (% gross total.)
1. *Discharged home* before or after 17 days in the ward	25·4	24·3	288	25·0
2. *Voluntary patients:*				
a. To mental hospitals	10·6	10·5	122	10·6
b. To the Maudsley Hospital	4·5	4·2	51	4·4
3. Mental after-care convalescent homes	3·0	2·9	34	3·0
4. Temporary patients	0·3	2·1	13	1·1
5. Certified (99% L.A. 1% Mental Deficiency Acts)	27·9	28·2	324	28·0
6. Senile: (*a*) to Tooting Bec Hospital (for senile cases); (*b*) died in the observation ward	11·2 / 5·6	10·8 / 5·8	128 / 66	11·0 / 5·7
7. To acute or chronic sick wards (of the general hospital and institution)	8·4	8·3	97	8·3
8. Died (not senile)	2·8	2·5	31	2·6

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(1) It will be noticed that some remained in the ward on a voluntary basis for a longer period than the seventeen days (limited to a further fortnight, except in very special circumstances, otherwise the congestion would have become unmanageable.

Most of these patients (105 out of 288 discharges) were fit to return to their usual routine, while some attended the Maudsley Out-Patient Department. If they had gone on to mental hospitals the percentage of voluntary patients would have been greatly raised.

Among the reasons for remaining in the observation ward, as an alternative to voluntary treatment in a mental hospital, were (1) that men might lose their work if they did not quickly return; (2) certain of the relatives influenced patients against going to a mental hospital; (3) to other patients the name "mental hospital" signified something which they felt applied to cases worse than themselves.

Also included in this section are several cases discharged to institutions or hostels whose relatives would not have them back, or who had previously lived alone, but who now needed supervision.

(2) It is considered that the most suitable types of voluntary patients were:

a. Minor recoverable psychoses and neuroses.

b. Major recoverable cases, particularly melancholics.

c. Docile schizophrenics.

d. Chronic neurotics.

Many patients in the first two groups were suitable for admission to the Maudsley Hospital.

It has been said that anyone who is certifiable but willing to go as a voluntary patient is suitable for such treatment, but all such cases cannot be recommended. For instance, some with a bad history and outlook should not be encouraged to go as voluntary patients, since in all probability they would not stay long enough to improve. This is not always appreciated by Justices. Those belonging to the types we believe to be "justifiably certifiable" were usually certified; in some of these cases it was difficult to secure enough satisfactory evidence of insanity during their stay in the ward, and a few had to be released to their protesting relatives. On the other hand, a few apparently suitable voluntary patients were returned within a few weeks recommended for certification; these were usually schizoid cases of whom the worst had not been seen.

(3) This group contains those patients unfitted on the one hand to go to ordinary convalescent homes, and on the other not needing mental hospital treatment or a further period in the observation ward. The Mental After-Care Association's convalescent homes are particularly useful for depressive cases who have recovered in the ward, perhaps after a suicidal attempt, and hence need more freedom under some supervision before returning to their normal duties.

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(4) Our figures for the cases sent as temporary patients show a considerable difference; this is because we set out to test entirely different experimental ideas on the Act. In the light of recent discussion on this matter the results may be of interest:

(*a*) In 638 cases one of us estimates that 23 acute non-volitional patients with fairly reasonable prospects of early recovery, might have been sent as temporary patients within the first week of admission. They were, however, kept in the ward instead of being dealt with at once under Sect. 5 of the Mental Treatment Act. It is of interest to note what followed. Of these 23 patients, 10 were returned home fully recovered within two to six weeks; 10 became voluntary patients at the mental hospitals, 2 were certified, and 1 died from sudden cardiac failure. Other confusional and non-volitional cases were numerous, but quite unsuitable to be temporary patients. These were certified, because of a bad previous history of mental abnormality, particularly if long-standing and unremitting, or perhaps of a "difficult", dangerous or unreliable type, or if markedly schizophrenic, paraphrenic or paranoidal; again this was the best procedure in the presence of progressive or irrecoverable organic disease, including the pre-senile degenerations, of which there were many amongst the confusional and maniacal cases admitted. Briefly those who were not likely to be discharged or become reliably co-operative after a reasonable period or treatment in a mental hospital, whether regaining volition or not, were considered to be unsuitable as temporary patients. Two entirely suitable cases were sent away under this section of the Act.

(*b*) In the 518 cases of the other writer, an attempt was made to send every strictly non-volitional case away as a temporary patient providing (i) his chance of recovery was good, and (ii) he was not expected to become volitional for some weeks. Only 11 such cases were found. Two are still temporary patients, 6 have recovered (in 11, 9, 5, 5, 2, 1 months respectively). Two became voluntary patients (one of these is almost fit to be discharged, and the other left, but was later certified). The eleventh was certified after two months.

Two conclusions may perhaps be drawn from these results: the first that an extraordinarily small number of patients are really suitable for temporary treatment unless this section of the Act is stretched beyond its clear legal meaning; secondly that after two or three weeks of treatment in an observation ward the percentage of cases for which the Act, strictly applied, can be used is negligible. However, since they either recover, or go away as voluntary patients, it does not seem to matter much.

(5) The figure of 28% for certification is in our estimation still rather high. It would be considerably and justifiably reduced if further use could be made of the observation wards as treatment centres, and if one further detention order beyond the usual fourteen or seventeen days could be made, say, on the consultant's written advice, in non-co-operative but nevertheless recoverable cases.

The Board of Control Report (1935) states that hopes are held that eventually only one-third of all cases admitted to mental hospitals will be certified.

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It is interesting to find that in a previous year, but after the Mental Treatment Act was passed, for a series of 212 consecutive admissions to the wards, 55% of males and 58% of females were certified. Certain types are probably always best certified; among these are patients with a bad social history or criminal tendencies, paranoid, schizoid or dementing cases who seem likely to be permanent and are not amenable to voluntary treatment; mental defectives with a superadded psychosis, dangerous senile cases, and some with organic disorders refusing voluntary treatment.

The above statement from the Board of Control's Report seems to be utterly inconsistent with the view also emanating from the same source, indicating that not enough use is made of the Urgency Order (extended to rate-aided patients by the 1930 Act). This Order obviously applies to the very acute cases, yet it seems quite clear that these are the cases that often quickly recover in an observation ward, and consequently have no need of mental hospital case.

A second point is that if it is decided to transfer these cases to mental hospitals, they are the very patients who provide most of the suitable temporary patients and have no need of certification.

Thirdly, the Board of Control's Report states the purpose of the observation wards to be the diagnosis of difficult cases. Are not these the patients who provide the greatest difficulty to those not often seeing such types? Moreover they are the most likely to be confounded with organic disorders unless given the opportunity for hospital investigation. Those patients to whom the observation wards can give the greatest assistance are unnecessarily side-tracked by using the Urgency Order.

It would seem clear then that the observation ward is the essential unit in the present system, not only for reducing the numbers certified, but for avoiding their transfer altogether, which is a far more important matter from the patient's point of view.

(6) Nearly a third of the total senile admissions died, some of them within a short time of coming into the wards. The removal from home or hospital seemed unnecessary in some cases; on the other hand this might well have been due to the fact that they quickly settle down under the observation ward routine.

At present they hamper the true work of an observation ward, and it is to be hoped that it may be possible at some future date to send them directly from hospitals or their homes to the special hospitals for seniles, and that some such institution or part of the present ones may take the troublesome and dangerous types.

The Maudsley Hospital Medical Superintendent's Report states that "the rising prestige of the mental observation units is producing great changes, which will grow further when it becomes possible to clear them of the senile cases which now so greatly reduce their capacity". Observation wards are

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unnecessary for the diagnosis and disposal of most of these cases, and they are unsuitable companions for young psychotics, and especially neurotics.

(7) Reference to this considerable percentage of acute and chronic sick cases is made in the next section.

Section V.

Observation wards are in their infancy so far as their developmental possibilities are concerned—in fact we are still in the process of deciding what their purpose should be.

Returning to the Board of Control's Report, it is stated that the main purpose of observation wards is the diagnosis of doubtful cases, but that they have dangers in that active treatment may be encouraged. We hope this account of the work in an observation ward has shown that it is possible to avoid sending large numbers to the mental hospitals without trespassing upon those cases for which they " are the only places able to provide the specialized experience and the therapeutic resources necessary for successful treatment ".

In order to do this, however, we feel that, in certain cases, active treatment up to that point is to be encouraged, and that in fairness to the patient, it should be practised whilst the diagnosis of difficult cases is proceeding.

The second point from the Board of Control's Report concerning the purpose of the observation wards states: " Once it is clear that the patient requires treatment for mental illness, no time should be lost in transferring him to the mental hospital." Incidentally a point to be considered is that if one is to transfer patients rapidly, upon whom is the onus of the defence to rest if actions are brought for wrongful certification ? We have already stated that it seems unreasonable to hasten some patients' departure to a mental hospital under certificate, when they happen, at the time, to be unwilling to go as voluntary patients. They may often be made to realize the need of treatment within the next fortnight, or may even be able to return home within or soon after their seventeen days' detention. It is a waste of time and bad therapy to suggest to a patient that he may be certified if he does not go to a mental hospital as a voluntary patient, for he will generally agree to give nominal consent in order to effect his early discharge from the mental hospital. Prognosis is an essential factor in making a reasonable classification. The method of saying, " Will you go as a voluntary patient ? " and making him a voluntary, temporary or certified case according to whether he replies " Yes ", gives no answer, or " No ", is not as simple as it sounds. In nearly every case the patient requires time for investigations to be made, and this is where the view of the observation ward as being merely a sorting and clearing station breaks down.

Another reason for delaying the transfer is found in the number of cases later passed on to acute or chronic sick wards. 8·3% of 1,156 patients were thus removed after recovery from a transient psychosis. Had there been no

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observation ward these cases would all have gone to mental hospitals, mainly under certificate.

We are now left in the rather awkward position of having to state what conclusions may be drawn as to the purpose of the wards.

Two distinct types of case come into an observation ward. The first group contains the "justifiably certifiable" patients who should be transferred under the Lunacy Acts as soon as grounds for certification can be found; and "obvious" voluntary and temporary cases whose papers should be completed as rapidly as possible. This is as suggested in the Board of Control's Report.

The second and much larger group needs investigation, and should, we believe, be to some extent actively treated. Some will recover and go home before seventeen days, or after a short extra period on a voluntary basis in the ward. Others will improve sufficiently to be made into good voluntary patients. A number will settle down from a transient psychosis sufficiently to go to general wards for the treatment of their physical illness. The institutional cases and social "problems" have to be disposed of individually as thought fit, though in the future systems will probably be evolved for dealing adequately with these cases. Finally a greatly reduced number eventually need certification under the Lunacy Act.

The observation wards provide ideal subjects for teaching purposes. In the Maudsley Hospital Medical Superintendent's Report it is stated that the need of acute cases for study could, in the future, be met largely by the observation ward, in which the cases are of such a kind that experience of them cannot reasonably be omitted from either undergraduate or post-graduate training in psychiatry.

Lastly the observation ward has a purpose in providing excellent research material; and an opportunity is here available for the treatment of cases at their onset.

Few observation wards in other counties have consultant psychiatrists, officers and staff experienced in mental diseases, and all prognostic aids, including social histories, although some are placed conveniently near to a general hospital. There are many practitioners in all parts of the country who would welcome the facilities of such a unit within easy reach; and the responsibility of disposal, especially of certification, could be borne by officers under the local authority. This would diminish overcrowding in the mental hospitals, increase the percentage of voluntary and temporary patients, and add a useful and necessary link in the organization of the mental health service.

The greater the number of relatives and patients that one sees, the more certain does one feel that the whole outlook on mental disorder will have to undergo great changes before it can be fair to transfer or advise the voluntary admission of a patient to a mental hospital, unless this is absolutely essential. The form of admission does not matter nearly so much as the fact that he goes

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there, even though "mental" is omitted from the name. There can be no doubt, however, that the patient's future life is less affected by voluntary or temporary treatment than by certification. The re-naming is a step forward, but there are far more important factors to be considered.

A fundamental point in the relatives' outlook is that the patient is going to a hospital where the very great majority of its occupants are destined to stay for life. This is so well known that even the admission villas are inseparably attached to the main hospital in the minds of the public. Again, the London County Council have progressed by devoting a hospital to recoverable cases, other than those voluntary patients going to the Maudsley, though one can imagine that it may be difficult to persuade them to go elsewhere when this becomes generally known.

The ultimate solution must depend largely upon general education as to the nature of the hospitals and treatment, the form of mental disorder, and its regard in the same light as other illness. From this point of view it is instructive to compare the outlook on tuberculosis or cancer. What, we wonder, would be the public reaction to a pseudo-phthisical comedian who made the sanatorium a subject of humour, or who became witty at the expense of a fungating carcinoma? Yet nearly every music-hall programme contains its modern counterpart of the traditional fool.

In the present system of mental treatment it is the observation wards that have the closest contact with the relatives; and it is here that the patient himself makes, or has made for him, a plan for his welfare, which will influence the whole of his future life. Since the observation ward is the smallest and consequently the most flexible portion of the system, it must adjust its working to the requirements of those for whom the complete organization has been evolved, and from this basis nothing but improvement, understanding and smoother co-operation can result.

Summary.

The work in an observation ward which deals with 2·5% of all rate-aided patients admitted to county and borough mental hospitals (which number 23,000 per annum) is described.

The types of cases entering the ward are classified and discussed. The various alternative methods of disposal are considered, in particular Section 5 of the Mental Treatment Act and Urgency Orders, both in their application and value; the figures are analysed.

The writers state that they believe the observation wards to be a useful and essential part of the system of dealing with mental disorder. Their purpose and value are examined with special reference to the Annual Report of the Board of Control for 1935 (Part I) and the Maudsley Hospital Medical Superintendent's Report (1932-35).

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We wish to acknowledge the very kind advice of the Chief Officer of the L.C.C. Mental Hospitals Department, Mr. R. H. Curtis, and of Dr. Letitia Fairfield, both of whom have read the paper.

We are also grateful to the Medical Superintendents of the London County Council hospitals who have sent us details of the progress of cases. In particular we would like to thank Dr. O. W. Roberts, the Medical Superintendent, for allowing us to use the material, and for his encouragement; and Prof. Mapother for his kindly criticism.

We wish to express our indebtedness to Sir Frederick Menzies, since the work was done under the Public Health Department, and to state, in accordance with Order 304, that the London County Council can accept no responsibility for any of the opinions expressed in this paper.

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"TEMPORARY" TREATMENT: AN ANALYSIS OF THIRTY CASES.

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SINCE the passing of the Mental Treatment Act, 1930, 30 patients have been admitted to St. Ebba's Hospital under Section 5, and it is proposed to analyse the type of case admitted, and to mention some of the difficulties encountered in treating patients on a temporary basis.

Of the 30 patients, 25 were female and 5 male, and the following diagnoses were made:

	Female.	Male.
Schizophrenia	17	3
Toxic confusional states	3	..
G.P.I.	2	..
Cerebral arteriosclerosis	1	1
Delirium tremens	..	1
Delirious state.	1	..
Alcoholic Korsakov syndrome	1	..

CLINICAL MATERIAL.

As will be seen, 20 patients were suffering from schizophrenia, 17 of whom were in a state of acute catatonic excitement, while 3 were mute and stuporose. The former were restless, excited, hallucinated, disjointed in talk and suffered from bizarre delusions and passivity feelings.

All these patients, with one exception, a woman of 40 with a late schizophrenic or paraphrenic state, could be looked upon favourably from the prognostic point of view, as the previous history revealed good personalities and abrupt onset of the illness, which was of short duration before admission. The duration varied from five days to three months, the average time being two months. The average age was 32, the oldest being 37 and the youngest 16. Most of the patients, in addition, showed adequate precipitating factors, such as the puerperium, physical ill-health or adequate mental stress.

The toxic-confusional states were the result of influenza in two cases and tonsillitis in one, and were abrupt in onset, as were the cases of delirium tremens and delirium. The patient suffering from the Korsakov syndrome showed

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gross memory impairment with confabulations, and was definitely non-volitional. The two cases of G.P.I. were admitted as toxic-confusional cases and were diagnosed as G.P.I. after admission, but as all cases of G.P.I. in the L.C.C. mental hospitals are treated at Horton Hospital, they were not suitable cases for retention in St. Ebba's Hospital.

The two patients suffering from cerebral arteriosclerosis were not suitable for admission on a temporary basis, owing to the prognosis, and were the only ones which should not have been made " temporary " patients, but certified in the ordinary way.

RESULTS.

	Female.				Male.			
	Rec.	Rel.	N.I.	Died.	Rec.	Rel.	N.I.	Died.
Schizophrenia	6	3	8	..	2	1	..	..
Toxic-confusional states	1	1	..	1	..	..	..	..
G.P.I.	..	..	1	1	..	..	..	..
Cerebral arteriosclerosis	..	..	1	..	..	1	..	..
Delirium tremens	..	..	..	..	..	..	..	1
Delirious state	..	..	..	1	..	..	..	..
Korsakov syndrome	..	1	..	..	..	..	..	..
Total	7	5	10	3	2	2	..	1

It will be seen from the table that—

9 patients were discharged " recovered ".

7 " " " " relieved " (of whom 3 remain in the hospital on a voluntary basis).

10 are " not improved " (of whom 6 are still in hospital, 1 was transferred as a temporary patient to another hospital, and 3 were discharged to observation wards, and are now certified in other mental hospitals).

4 died.

It is now proposed to analyse these results in detail.

RECOVERED GROUP.

Female schizophrenics.

	Recovering volition on admission.	Time of becoming V.P. after admission.	Duration of Stay as V.P.
1.	Yes	28 days	6 months.
2.	No	5½ months	3½ "
3.	"	5 weeks	Still in hospital.
4.	"	4 months	5 weeks.
5.	"	5 "	Still in hospital.
6.	"	7½ "	" "

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Female confusional.

	Recovering volition on admission.	Time of becoming V.P. after admission.	Duration of Stay as V.P.
7.	Yes	36 days	Left same day to convalesce in country.

Male schizophrenics.

8.	Yes	7 weeks	Still in hospital.
9.	„	28 days	6 months.

It will be noticed that four patients are still in hospital on a voluntary basis. These patients have made good recoveries, but have not yet departed, as they have been advised to remain until some environmental difficulties have been removed.

Relieved Group.

Females: Schizophrenics.

10. This patient recovered volition 3½ months after admission, refused to stay on a voluntary basis, and, as she was a private patient, was taken out by her husband under Section 72, 21 days after recovering volition.

11. This patient began to recover volition towards the end of her 6 months' stay, and as she refused to stay on a voluntary basis she was discharged to her sister's care at the expiration of her order by operation of the law.

12. This woman was recovering volition on admission, was made a voluntary patient 32 days after, and remains in hospital as a voluntary patient, as she is still rather aloof, detached and abstracted, though volitional.

Confusional state.

13. This patient became volitional 25 days after admission, was made a voluntary patient, but departed 3 weeks later against advice.

Korsakov syndrome.

14. This patient became volitional 5 months after admission, became a voluntary patient, and is still in hospital, as she is paranoid, facile and irritable, and shows some organic deterioration.

Males.

15. This patient, suffering from schizophrenia, was recovering volition on admission, became a voluntary patient 28 days later and left 2 days later

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on his father's advice. He has since been readmitted on a voluntary basis, and is still in hospital.

16. This man was admitted in a confusional state as a result of cerebral arteriosclerosis, and became volitional 4½ months later. He agreed to stay on as a voluntary patient and remains in hospital, still in a state of depression with signs of organic deterioration.

Not Improved Group.

17, 18 and 19. These three patients have only been in hospital for a short time, and are still in a state of schizophrenic stupor.

20. This patient has been in hospital since October 22, 1936, and her temporary order was extended for a further 3 months on March 25, 1937. Her order is being extended again for a final 3 months, and although she has improved somewhat and the prognosis appears to be good, it is doubtful if she will have recovered volition at the end of her year, and certification may be necessary.

21. This patient recovered volition one month after admission, but 20 days later was again non-volitional. She has been in hospital for 4 months, and as the prognosis appears to be good, the order will be extended if necessary.

22. This patient became volitional one month after admission, and became a voluntary patient. One month later she became restless, excited and violent, gave in her notice to leave hospital and was sent to an observation ward.

23. This patient, whose prognosis was good, had been a temporary patient for 9 months, and as she was unlikely to recover in another 3 months she was discharged to an observation ward, where she was certified and sent to another mental hospital.

24. This patient was admitted as a voluntary patient, and as she became non-volitional 3 months after admission she was made a temporary patient. She improved, and became volitional 3 months later, but refused to stay in hospital as a voluntary patient, and was discharged by operation of the law, at the end of 28 days after recovering volition, to an observation ward.

25. This patient was suffering from G.P.I., and was transferred as a temporary patient to Horton Hospital for treatment.

26. This patient has only been in hospital for 2 months, but as she is suffering from cerebral arteriosclerosis with a poor prognosis the order will probably not be renewed.

Deaths.

Four patients died from pneumonia, one of whom was suffering from G.P.I., one from a toxic confusional, and two from delirious states. All died within 11 days of admission, and exhausted themselves as a result of their restlessness.

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COMMENTS.

There are certain points which arise in dealing with patients under Section 5, and the first is the matter of prognosis. It is obviously no use selecting cases for treatment on a temporary basis, even though they are non-volitional, where the prognosis is bad, since eventually they must be discharged and certified. Thus it is important to have the cases examined in the first place by competent psychiatrists. Another important point is that the total time over which the order can be extended appears to be too short. Thus patient 23 had to be certified, as she was unlikely to get well in 12 months, but in all probability will make an excellent recovery in two years; while patient 20, whose prognosis also appears to be good, will also require certification if she is still non-volitional at the end of a year. It is really in this type of case, together with the so-called "perplexity" states seen in schizophrenia and depression, which have a long duration but a good prognosis, that temporary treatment would be of value, and yet the Act defeats itself by not allowing the patient a sufficiently reasonable time in which to recover volition. There appears to be no good reason why temporary treatment should not be extended up to 2 years.

Another difficulty arises as to when a patient is to be considered volitional. For instance the first time a patient says that he wishes to go home or stay in hospital, is he to be considered volitional? Many patients in a state of stupor have periods of accessibility, and then lapse again into stupor. This means that periodically slips have to be sent in to the Clerk of the Hospital, stating that the patient has again become volitional or non-volitional. There appears to be no hard and fast rule as to when this must be done, but it would be more practical to wait until the twenty-eighth day from the day the patient became either volitional or non-volitional to decide whether he was *i.s.q.*, except for the fact that alternative treatment has to be arranged by the twenty-eighth day. This consists in the making of the patient into a voluntary patient, if he is volitional and willing to stay in hospital, or to reversion to temporary status if he is non-volitional, and this requires some time. This period of 28 days is therefore in practice too short, and should be increased to at least 2 months. Barbour (1) also found that this period of 28 days was too short for practical purposes. In addition to the above difficulty, the patient at times refuses to remain in hospital after recovering volition; he goes home, relapses, and requires certification.

Thus patient 15 left two days after becoming a voluntary patient, and has since returned to hospital as a voluntary patient for further treatment. If the 28 days were increased to 2 months the additional month may make a profound difference to the patient's health before leaving, and often having emerged from a state of stupor or confusion, during the extra month he may come to regard the hospital from a different point of view and be willing to remain on a voluntary basis.

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Again many patients are recovering volition on admission, and if unwilling to stay on a voluntary basis would have to be discharged in a month's time. This is much too soon, and often means a relapse with readmission to hospital, which is surely detrimental to the patient. Marshall (2) states that only 10% of patients who are suitable for treatment on a temporary basis are fit for discharge in this time, and even 10% is probably high.

It appears, then, that Section 5 of the Mental Treatment Act, 1930, is of value in those psychoses with a good prognosis, and where volition is absent, but that modifications are necessary if full benefit is to be derived from its application.

References.—(1) Barbour, W. J., "Temporary Treatment in Mental Hospital Practice", *Brit. Med. Journ.*, Feb. 6, 1937, p. 281.—(2) Marshall, J. K., "A Note on the Potential Use of Temporary Treatment", *Journ. Med. Sci.*, Jan., 1936, lxxxii, No. 336, p. 43.

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ACETARSOL IN THE TREATMENT OF LATE CONGENITAL SYPHILIS AMONGST MENTAL DEFECTIVES.

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THE treatment of congenital syphilis, in spite of the advances in the general treatment of syphilis in the last thirty years, still leaves much to be desired. Veeder, after treating some 500 cases of congenital syphilis in St. Louis between 1912 and 1920, came to the conclusion that the results of treatment were most unsatisfactory, and that very little was to be gained by the treatment of the syphilitic child. He advocated the treatment of the parents to prevent the birth of syphilitic children. Nabarro is of opinion that the criterion of cure should be the complete cessation of any symptoms of activity and a normal blood and cerebro-spinal fluid. He has not been able to fix any limit of time after which he can regard the patient as cured. He mentions a case of his which relapsed with interstitial keratitis after having had a negative Wassermann reaction for eight years. Ambrose King doubts whether congenital syphilis is ever really cured; he states that "cases had been seen in which manifestations of congenital syphilis appeared only after the lapse of a number of years, and others in which active lesions occurred, but with a negative Wassermann".

At Caterham Hospital, where approximately 10% of mentally defective children were found to be heredo-syphilitics, many had received specific treatment, but the final results could only be described as poor. In 1933 I reported a case of a boy with congenital syphilis who had received prolonged treatment from an early age, in spite of which he developed meningo-vascular syphilis with hydrocephalus and interstitial keratitis. He died at the age of 17, and a post-mortem examination showed widespread lesions attributable to congenital syphilis.

In institutions where large numbers of patients are treated, ease of administration of drugs is desirable. In this respect acetarsol, which is administered by mouth, has obvious advantages over the arseno-benzene derivatives given by injection. Children dread the weekly pricking, which may easily cause psychological upset. For this reason, if for no other, treatment by an oral preparation of arsenic is to be welcomed, if results are at all comparable.

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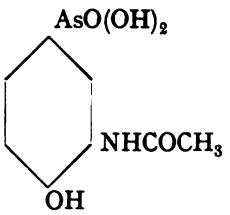
COMPOSITION, PROPERTIES AND HISTORY OF DRUG.

Acetarsol (B.P. Addendum 1936), also known as orarsan, stovarsol, spirocid, osarol and acetarsone, is 3 acetyl-amino 4 hydroxyphenyl arsonic acid, and is represented by the following graphic formula :



It belongs to the pentavalent series of arseno-benzene derivatives, being allied to the better-known tryparsamide. Of a white amorphous appearance, it is practically tasteless and almost insoluble in water, but somewhat soluble in alkaline solutions. Its arsenical content is 27·2%, which compares with 20% in neosalvarsan and 32% in salvarsan. Originally prepared by Ehrlich in 1909 (preparation 594), it was discarded after trial as being too toxic. In 1921 the drug was examined again by Fourneau and his co-workers and renamed " Fourneau 190 ". Levaditi and Nabarro-Martin in 1922 reported that monkeys and rabbits with primary sores were cured by stovarsol administered orally, following which 80 syphilitic patients were treated. In 30 with primary syphilis the chancre disappeared in from 5 to 15 days. Good results were also obtained in secondary and tertiary lesions, but probably owing to the short period of treatment (about 15 days), there were frequent relapses. Sézary and Barbé treated 125 unselected cases of general paralysis (1921–1929), and obtained good results in those with mania and expansive delusions. In 34 of this type 17 were able to resume work and 4 derived benefit. Serological reactions were improved in 34% of total cases.

MacQueen found that spirocid had many advantages over N.A.B. in the treatment of infected Arabs in Palestine.

Stovarsol has also been used in the treatment of yaws, malaria and amœbiasis, but it has been used most successfully in congenital syphilis. Maxwell and Glaser, Abt and Traisman all report very favourably on the action of this drug in congenital syphilis. Nabarro used it in the treatment of a number of congenitally syphilitic infants in residence at St. Margaret's Hospital, several of whom had become serologically negative. It was, however, too early to say whether they were cured. He states, " If it should turn out eventually that stovarsol can cure congenital syphilis, this will be a very distinct advance over treatment by injections ". The plan of dosage he used was that outlined by Prof. Hamburger and his assistant, Dr. Alois Bratusch-Marrain, who is of opinion that the treatment of congenital syphilis with

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spirocid is superior to that with neosalvarsan. The Wassermann reaction disappears more quickly and there is better intellectual development. In prolonged treatment it is easier of administration, especially to out-patients. Given in the following manner, toxic reactions due to any hypersensitiveness to the drug are minimized:

For the first 7 days,	0·005 grm.	(gr. $\frac{1}{12}$)	per kgrm.	body-weight	daily.
,, following 7 days	0·01 ,,	(gr. $\frac{1}{6}$)	,,	,,	,,
,, ,, ,,	0·015 ,,	(gr. $\frac{1}{4}$)	,,	,,	,,
,, ,, 42 days	0·02 ,,	(gr. $\frac{1}{3}$)	,,	,,	,,

There is then an interval of 4 to 6 weeks, followed by other similar courses until the Wassermann reaction has become negative three times. Even then it is recommended that further half-yearly courses be given for safety. Davidson and Birt, following the method recommended by Maxwell and Glaser, report excellent results in the treatment of congenital syphilis with stovarsol at the Out-patients' Department of the Winnipeg Children's Hospital. Of 27 patients who had been treated for over 7 years by other methods, only 6 Wassermann reactions became negative as compared with 14 changed from positive to negative, and 2 reduced to doubtful in 2 years in a group of 23 treated with stovarsol. They claim that "the decided increase in the percentage of cases cured is enough in itself to assure this drug a permanent place in the therapy of congenital syphilis".

Treatment of Congenital Syphilis at Caterham Hospital (L.C.C.).

In the spring of 1934 it was decided to treat with acetarsol a heterogeneous group of mental defectives suffering from late congenital syphilis. The diagnosis of congenital syphilis was based on (1) family history with, in some instances, blood Wassermann reaction of the mother, (2) physical signs, and (3) serological tests.

The group consisted of 29 cases of both sexes and all ages. As far as known, the majority had not received any prior specific treatment. The duration of treatment was about 2¼ years. A control group of 12 other cases received no treatment during that time.

For convenience in analysis the 29 cases are divided into two groups—(1) those with normal cerebro-spinal fluids, and (2) those with abnormal cerebro-spinal fluids, hereafter referred to as Groups A and B respectively. The control group is designated Group C.

Dosage.

The scheme of dosage was mainly that recommended by Bratusch-Marrain, with modifications to suit individual cases. For instance, it was found inadvisable for the maximum dose to exceed gr. 5 (grm. 0·33) *t.d.s.* A few cases were treated with minimal doses only. The daily dose was split into

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approximately three equal parts, these being administered crushed in water, half an hour before the principal meals.

Experimental Observations.

When taken orally, does acetarsol reach the blood and cerebro-spinal fluid; if so, in what concentration? This was answered by direct experiment. Through the kind co-operation of Dr. S. A. Mann, of the Central Laboratory of the L.C.C. mental hospitals, and of Dr. J. J. Fox, of the Government Laboratory, to both of whom I am indebted, the arsenical content of samples of blood and cerebro-spinal fluid of Case No. 24 was determined.

Case No. 24.—H. V. B—, male, æt. 27. Family history: 4th child of 8. Two were born dead, 2 died in early infancy, and 1 has recently been admitted to a mental hospital suffering from juvenile general paralysis.

Signs: Frontal bossing, hypoplastic teeth, absent xiphoid appendix. Spastic right hemiplegia. Pupils react to light poorly. Marked tremor of tongue and lips. Ataxia. Slurred speech.

He has been showing slow mental and physical deterioration for the last 10 years and is a typical example of juvenile general paralysis.

The experiment was carried out between the 3rd and 4th courses of acetarsol, when his body-weight was 49 kilos. The first specimen of blood and cerebro-spinal fluid was taken in the morning. Gr. 20 (grm. 1·3) of acetarsol was then given orally, crushed in water, on an empty stomach. Further samples of blood and cerebro-spinal fluid were then taken at the following times with the appended results:

Sample.	Time.	As_4O_6 parts per million.
1	Cerebro-spinal fluid 10.40 a.m.	Less than 0·4.
2	,, 11.50 a.m.	,, ,, 0·4.
3	,, 12.50 p.m.	Approximately 0·4.
4	,, 2.0 p.m.	Less than 0·4.
5	,, 4.30 p.m.	Approximately 1.
6	,, 10.40 a.m. (next day)	Less than 0·4.
1	Blood from arm vein 10.40 a.m.	0·9
2	,, ,, 11.50 a.m.	1·8
3	,, ,, 12.50 p.m.	3·5
4	,, ,, 2.0 p.m.	5·2
5	,, ,, 4.30 p.m.	9·3
6	,, ,, 10.40 a.m. (next day)	21·1

These findings would appear to indicate non-penetration of the drug into the cerebro-spinal fluid. The rise in the 4.30 p.m. specimen was due to slight blood contamination. The arsenical content of the systemic blood on the

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other hand rises from 0·9 to 21·1 parts per million in 24 hours, clearly showing absorption from the alimentary tract.

Serological Reactions.

These were carried out at the Central Pathological Laboratory of the London County Mental Hospitals. Specimens of blood and cerebro-spinal fluid were examined before and at the end of every course. The blood-serum was examined by the Wassermann reaction and the macro-Meinicke clarification reaction (M.K.R.). The cerebro-spinal fluid was examined for increase of cells and protein and the Wassermann reaction and Lange's colloidal gold test were done. The technique used was that of Mann and Partner.

Briefly, for the Wassermann reactions, the figure following the first plus indicates the number of units of complement (1 unit equals approximately 0·01 c.c. of pure guinea-pig serum) absorbed by 1 c.c. blood-serum or cerebro-spinal fluid. The plus following the figure indicates that a further absorption of complement would take place if the range of dilution of serum or cerebro-spinal fluid were extended.

For the macro-Meinicke clarification test (M.K.R.) four tubes are set up. A positive reaction is indicated by complete clarification of at least one tube.

Group A.

This consisted of 19 cases (5 males and 14 females); 12 of these were under 26 years, and 7 over. Case No. 19, a female aet. 48, had active lesions but negative serology. Of 17 cases showing positive Wassermann reactions prior to treatment, 8 became negative, 7 were reduced in strength, and 2 were unchanged after treatment. The M.K.R. reaction was more resistant; of 18 positives, 6 became negative, 6 were reduced in strength, and 6 were unchanged. The corresponding figures for the control group (C) were: Wassermann reactions positive, 7; became negative, 1; reduced in strength, 0; unchanged, 4. M.K.R. positive, 10; became negative, 1; reduced in strength, 2; unchanged, 7.

In Group A there was no increase of positivity after treatment, but in Group C over approximately the same time, 4 negative Wassermann reactions and one doubtful became positive, 2 increased in strength. Though the control group is not exactly comparable in respect of age and sex to the treated group, the difference in the final serological reactions is so marked as to leave little doubt as to the drug's influence.

It will be noted that as many as 5 out of 6 who were finally both Wassermann and Meinicke negative were under 20 years of age. This would appear to indicate better serological response to treatment in the younger members of this group.

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TABLE I.—*Group A : Cases with Normal Cerebro-spinal Fluid before, during and after Treatment.*

Case No.	Sex.	Age.	Grade.	Number of courses.	Before treatment. Blood.		After treatment. Blood.		Weight in kg.		Hb in grm. per 100 c.c. blood.		Highest eosinophil count. (%).	Toxic reactions.	Clinical results (improvement).		Remarks.
					W.R.	M.K.R.	W.R.	M.K.R.	Before treatment.	After treatment.	Before treatment.	After 3rd course.			Physical.	Mental.	
1	M.	12	Imb.	4	+15	++	—	—	34·5	45·4	12·8	12·0	23·4	..	Good	Good	..
2	,,	17	,,	6	—	++++	—	—	42·0	43·3	12·8	13·9	3·6	..	Moderate	*Nil*	..
3	,,	18	,,	6	+30^{+}	++++	+30^{+}	++++	48·6	54·2	13·1	12·3	6·3	..	Good	,,	..
4	,,	20	,,	7	+30^{+}	++++	—	+	46·4	48·6	13·3	13·2	3·4	..	,,	Moderate	..
5	,,	25	F.-m.	5	+6	++++	—	—	51·3	54·6	..	..	..	..	,,	Good	..
6	F.	20	Imb.	6	+30^{+}	++++	—	+++	55·9	56·2	11·8	12·8	2·0	Dermal	Very good	Moderate	Minimal dosage.
7	,,	30	,,	6	+30	++++	—	+++	37·7	39·0	12·8	14·2	3·0	,,	Moderate	,,	..
8	,,	14	,,	7	+30^{+}	++++	+15^{+}	+++	30·5	34·9	11·8	12·5	1·0	..	Good	,,	..
9	,,	33	F.-m.	6	+30^{+}	++++	+30	++++	58·0	60·7	..	..	..	..	,,	,,	Active signs.
10	,,	32	,,	7	+30^{+}	++++	+30^{+}	++++	60·5	62·6	10·2	12·3	2·0	Herpes	,,	,,	..
11	,,	31	,,	7	+15^{+}	++++	+6	+++	72·7	68·4	11·8	13·8	1·7	Lungs	Very good	Good	..
12	,,	14	Imb.	7	+30^{+}	++++	+15^{+}	++++	28·2	34·0	10·2	12·6	3·1	Dermal	,,	Moderate	Minimal dosage.
13	,,	17	,,	7	+30^{+}	++++	+15	+++	53·6	57·0	10·2	11·8	5·0	,,	Good	Good	..
14	,,	13	,,	5	+30^{+}	++++	—	—	46·8	39·0	12·8	13·0	3·0	Pharyngitis	,,	Moderate	..
15	,,	31	,,	7	+30^{+}	++++	+15^{+}	++++	60·5	64·0	12·8	13·0	1·0	Dermal	Very good	,,	..
16	,,	17	,,	7	+15^{+}	++++	—	—	54·3	53·1	11·6	12·6	0·6	,,	,,	Good	..
17	,,	59	,,	5	+30	++++	+6	++++	59·7	56·6	13·0	12·8	2·8	..	Died	..	Aplastic anæmia.
18	,,	16	,,	5	+30	++++	—	—	35·4	35·8	..	..	..	..	Good	Good	..
19	,,	48	,,	4	—	—	—	—	45·4	48·8	12·7	13·9	0·7	..	,,	Moderate	Active signs.

Imb. = Imbecile. F.-m. = Feeble-minded (Moron). W.R. = Wassermann reaction (+30^{+} = strongly positive; +6 = weakly positive).
M.K.R. = Macro-Meinicke clarification reaction (++++ = strongly positive).

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TABLE II.—*Group B: Cases with Abnormal Cerebro-spinal Fluid before Treatment.*

Case No.	Sex.	Age.	Grade.	Number of courses.	Time (before or after treatment).	Blood.		Cerebro-spinal fluid.				Weight in kgrm.	Hb. in grm. per 100 c.c. blood.	Highest eosinophil count. (%).	Toxic reactions.	Clinical results (improvement).		Remarks.
						W.R.	M.K.R.	W.R.	Cells.	Pandy.	Lange.					Physical.	Mental.	
20	M.	11	Imb.	7	Before	$+30^{+}$	++++	—	—	++	5555443210	27·5	13·3	3·0	Drowsy	Moderate	*Nil*	..
					After	+30	+++	+6	±	±	4433211000	24·5	12·3					
21	,,	11	,,	6	Before	$+30^{+}$	++++	$+30^{+}$	++	++	5555543210	22·3	11·6	7·0	Herpes	Good	Moderate	A case of juvenile G.P.
					After	$+30^{+}$	++++	+30	—	+	5554321000	17·2	12·8					
22	,,	12	,,	7	Before	$+30^{+}$	++++	+15	+	+	5544321000	24·8	11·8	2·7	..	Very good	Good	..
					After	$+30^{+}$	++++	+6	—	—	—	30·8	13·0					
23	,,	13	,,	6	Before	$+30^{+}$	++++	$+3^{+}$	+	+	4443321000	42·1	12·8	4·0	..	,,	,,	..
					After	—	—	—	—	—	—	42·2	12·8					
24	,,	26	,,	7	Before	—	+	—	—	+	3322110000	47·0	12·3	16·0	..	Moderate	*Nil*	A case of juvenile G.P.
					After	—	—	—	±	±	4433210000	50·0	14·4					
25	,,	16	,,	6	Before	$+30^{+}$	++++	+6	++	++	3344553210	28·4	12·3	4·0	..	Good	Moderate	..
					After	$+30^{+}$	++++	—	—	—	—	35·4	12·8					
26	F.	12	,,	7	Before	$+30^{+}$	++++	$+30^{+}$	+	++	5555554321	25·5	10·9	8·0	Dermal	Very good	,,	Minimal dosage.
					After	$+30^{+}$	++++	+3	—	±	3222110000	39·4	12·3					
27	,,	34	F.-m.	7	Before	$+30^{+}$	++++	+30	++	++	5555432100	56·3	12·8	3·7	..	Good	,,	..
					After	$+30^{+}$	++++	+15	+	++	5555432100	58·5	13·5					
28	,,	14	,,	3	Before	$+30^{+}$	++++	+15	±	±	2221110000	36·7	..	..	..	,,	,,	A case of juvenile G.P.
					After	$+30^{+}$	++++	+15	—	±	—	44·5	..					
29	,,	51	Imb.	7	Before	$+30^{+}$	++++	—	+	+	—	69·5	13·4	1·8	..	*Nil*	*Nil*	..
					After	$+30^{+}$	++++	—	—	—	—	59·8	13·9					

Cerebro-spinal fluid.—Cells negative (—) = under 6 per c.mm. Cells ± = 7 to 8 per c.mm. Cells + = 9 to 20 per c.mm. Cells ++ = 20 to 30 per c.mm. Cells +++ = 30 to 40 per c.mm. Cells ++++ = Over 40 per c.mm.

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TABLE III.—*Group C: Untreated Control Cases; Observations made at Times Roughly Corresponding to those of Group A.*

Case No.	Sex.	Age.	Grade.	1934. Blood.		1936. Blood.		Weight in kgrm.		Clinical results. (Improvement.)		Remarks.
				W.R.	M.K.R.	W.R.	M.K.R.	1934.	1936.	Physical.	Mental.	
C. 1	F.	20	F.-m.	±	+++	+30+	+++	63·5	67·5	*Nil*	*Nil*	..
C. 2	,,	48	Imb.	+30	++++	+30+	+++	50·8	51·3	Moderate	,,	..
C. 3	,,	44	,,	+30	++++	+30	++++	58·9	62·1	*Nil*	,,	..
C. 4	,,	20	,,	—	+++	+15	+++	46·3	51·7	,,	,,	Epileptic. Fits increased.
C. 5	,,	22	,,	+30+	++++	+30+	++++	44·5	43·6	,,	Moderate	..
C. 6	M.	10	,,	+30	+++	—	—	25·8	29·7	Good	Good	..
C. 7	,,	24	,,	+30+	++++	+30+	++++	47·7	47·2	*Nil*	*Nil*	Epileptic. Fits increased.
C. 8	,,	21	,,	—	++++	+10	+++	43·6	42·2	,,	,,	..
C. 9	,,	25	,,	+30	++++	+30	++++	51·2	53·5	Moderate	,,	Epileptic. Fits increased.
C. 10	,,	27	,,	+30	++++	+30+	++++	44·0	43·6	*Nil*	,,	..
C. 11	,,	27	,,	—	—	+10+	++	46·3	47·6	,,	,,	..
C. 12	,,	20	,,	—	—	+15	++++	49·5	50·8	Moderate	,,	..

Group B.

This comprised those with abnormal cerebro-spinal fluid. There were 10 cases, 6 males and 4 females. Included are 3 cases of juvenile general paralysis (Nos. 21, 24 and 28), the last of whom is still undergoing treatment; the others were considered to be examples of meningo-vascular syphilis.

Blood sera.—Of 9 with positive Wassermann reactions, only 1 became negative and 1 slightly decreased in strength. Two out of 10 positive M.K.R.'s became negative (one was only slightly positive before treatment), and 1 slightly reduced in strength. These results are in marked contrast with those of Group A.

The cerebro-spinal fluid: Wassermann reaction.—Of 7 positive before treatment, 2 became negative and 4 reduced in strength. One (No. 20), originally negative, became slightly positive after treatment.

Cells.—Of 7 showing abnormal increase of cells before treatment, 6 were back to within normal limits and 1 showed reduction after treatment. The cells were of the lymphocytic type.

Pandy's test.—Of 9 originally positive, 4 became negative, 3 almost negative, and 1 reduced in strength after treatment.

Lange's colloidal gold test.—Before treatment 9 gave positive curves of varying strength, mainly of the paretic type. At the end of treatment, 4 of these were completely negative, 3 weaker, 1 unchanged, and 1 slightly stronger.

Of the total 10 cases with abnormal cerebro-spinal fluids, 3 such fluids became completely normal and 7 improved after treatment.

No controls were available in this group.

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TABLE IV.—*Number of Cases showing Serological Changes during Treatment.*

Total number of cases 29.	Blood.		Cerebro-spinal fluid.			
	W.R.	M.K.R.	W.R.	Cells.	Pandy.	Lange.
Number of cases negative before treatment . .	3	1 .	22	22	20	20
,, ,, ,, after 1st course . .	8	3 .	21	24	26	21
,, ,, ,, ,, 2nd ,, . .	7	2 .	22	26	24	22
,, ,, ,, ,, 3rd ,, . .	6	4 .	23	25	24	24
,, ,, ,, ,, 4th ,, . .	10	4 .	24	26	26	25
,, ,, ,, ,, 5th ,, . .	12	6 .	23	26	26	24
,, ,, ,, ,, 6th ,, . .	10	8 .	23	29	25	25
,, ,, ,, ,, 7th ,, . .	12	9 .	23	28	27	24

Pandy ± reactions and ± cells (slight increase) have been taken as negative.

RELAPSES.

As will be seen from the above table, serological relapse during treatment was fairly common in those whose blood Wassermann reaction or M.K.R. had become negative. The best results were obtained in the 10 cases showing abnormal increase of cells in the cerebro-spinal fluid—after the 6th course cells were in every case within normal limits. At the end of the 7th course, however, one case had relapsed.

The following are brief details of those cases showing serological relapse during treatment:

CASE No. 1.—S. M—. After 1st course both W.R. and M.K.R. became negative. Four months later they were still negative. Seven months later blood W.R. + 30, M.K.R. +. He then had a 2nd course, at the end of which his blood W.R. was negative, M.K.R. +++. After 3rd course both W.R. and M.K.R. were negative and remained so after subsequent courses.

This case is an example of the uselessness of giving a single course.

CASE No. 2.—H. M—. Blood M.K.R. negative at end of 4th course. At end of 5th course M.K.R. ++. At end of 6th course M.K.R. negative.

CASE No. 4.—W. F—. Blood W.R. negative at end of 4th and 5th courses. At end of 6th course W.R. + 6. At end of 7th course W.R. negative again.

CASE No. 5.—S. J—. At end of 1st course blood W.R. negative. After 2nd course W.R. + 6. 3rd course W.R. negative, 4th course W.R. + 6, and finally after 5th course W.R. negative.

CASE No. 7.—M. C—. At end of 5th course blood W.R. negative. At end of 6th course W.R. + 30. At end of 7th course W.R. negative.

CASE No. 11.—M. C—. At end of 4th course blood W.R. negative. At end of 5th course W.R. + 6. At end of 6th course W.R. negative again.

CASE No. 16.—A. B—. At end of 2nd course blood W.R. negative. At end of 3rd W.R. + 6. At end of 4th and 5th courses W.R. negative. At end of 6th W.R. + 6, and finally at the end of the 7th course W.R. negative.

CASE No. 17.—A. T—. At end of 1st course blood W.R. negative. At end of 2nd W.R. + 30. At end of 3rd and 4th W.R. + 15, and at the end of the 5th course W.R. + 6. She died of aplastic anæmia soon after.

CASE No. 18.—R. G—. At end of 2nd course W.R. negative. At end of 3rd course W.R. + 6. At the end of 4th course W.R. + 30^{+}, and at the end of the 5th course W.R. negative. M.K.R. also negative for the first time.

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In contrast with the above the following two cases showed no signs of relapse after having become negative :

CASE No. 6.—J. C—. Blood became Wassermann negative at the end of the 4th course and showed no tendency to relapse subsequently.

CASE No. 14.—K. V—. From being Wassermann and Meinicke strongly positive before treatment, she became both Wassermann and Meinicke negative after the 1st course, and remained so throughout treatment, 4 further courses being given.

HÆMATOLOGY.

In 25 cases blood-counts were made before treatment and after the 1st and 3rd courses. Hæmoglobin content was estimated, using the Klett biocolorimeter with the Newcomer Standard. As a result of treatment, hæmoglobin was increased in 13 cases by 1 grm. or more per 100 c.c. blood, in 4 cases 2 grm. or more. Five cases showed loss of 1 grm. or less.

Red blood-cells.—21 cases showed gains of between 50,000 and 950,000; 6 of these gained over 500,000 per c.mm. Four cases showed losses of 20,000 to 210,000 per c.mm. There were increases in the colour index in 13 cases. Abnormal red cells were absent.

Leucocytes.—Counts varied from 6,000 to 12,000 per c.mm. In 8 cases the lymphocytic percentage was over 45. Treatment resulted in an increase in eosinophil cells in all 25 cases. Only 15 of these cases, however, showed an eosinophilia of 3% or over.

Urine.

Urinary examinations were made at least once a week throughout treatment. Abnormal constituents were uncommon.

CASE No. 13.—One of chronic nephritis, with albuminuria varying from 0·1 to 0·2% (Esbach), and moderate number of granular casts, showed no change during or after treatment.

CASE No. 17.—Developed mild glycosuria during the 2nd course, persisting throughout treatment.

CASE No. 27.—Showed transient albuminuria (1% Esbach) during the 2nd course.

CASE No. 29.—Showed slight albuminuria during the 2nd course.

CASES No. 12 AND 16.—Showed traces of reducing substance at irregular intervals during treatment.

Bile-salts or pigment were absent from all samples tested during treatment.

Weight.

Records of weight before and after each course were made. At the end of treatment 22 cases had gained, and 7 lost weight. In those of 20 years or under the average gain of weight per case was 4·2 kgrm. (14 cases), and the

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average loss 4·3 kgrm. (4 cases). Of those over 20 years old the corresponding figures were 2·7 kgrm. (8 cases) and 5·7 kgrm. (3 cases). With the exception of Case No. 29 all those showing loss of weight had at one time or another shown idiosyncrasy to the drug.

Two of the 3 cases of juvenile general paralysis gained weight.

Control group.—In those of 20 years or under the average gain was 3·6 kgrm. (4 cases). None lost weight. Of those over 20 the average gain per case was 1·8 kgrm. (4 cases), and the average loss 0·8 kgrm. (4 cases).

Epilepsy.

There were 8 epileptics in the treated and 3 in the control group. Fits were of the major type, non-Jacksonian in character, and, when numerous, bromides or luminal had been administered continuously. This has unfortunately introduced a factor whose influence has to be taken into account in the assessment of the value of acetarsol in epilepsy associated with congenital syphilis. Two further unselected small groups of non-syphilitic epileptics are therefore given for comparison, one treated by bromides, and the other by luminal.

Treated Group.

Case No. 5 had his first fit during 1934, prior to treatment with acetarsol. He had 1 fit in 1935 and none in 1936. No sedatives given.

Case No. 9 had 7 fits in 1934, 2 in 1935 and 2 in 1936 (up to June).

Case No. 12 had fits for the first time in February, 1935. She had 8 during that year and 4 up to July, 1936.

The 5 remaining cases had sufficiently numerous fits to allow comparison with the untreated and other groups.

The influence of acetarsol is best seen in cases Nos. 4 and 20, where a progressive reduction of fits occurred.

Case No. 14 did not show any real reduction of fits until 1936, when there was a sudden drop.

Case No. 17 showed a reduction of fits up to two months before death.

Case No. 10 was the only one to show an increase in fits, but the increase was small. It will be noted, however, that she was the only one in this group taking luminal.

Control group.—(Untreated congenital syphilitics with epilepsy). In this group the fits tended to increase in number, the administration of luminal appearing to have only a temporary sedative influence.

Is the reduction of fits in the acetarsol-treated group mainly due to the influence of bromides, and the increase in the control group to luminal? The answer is not easy, but I would suggest that the reduction of fits in the

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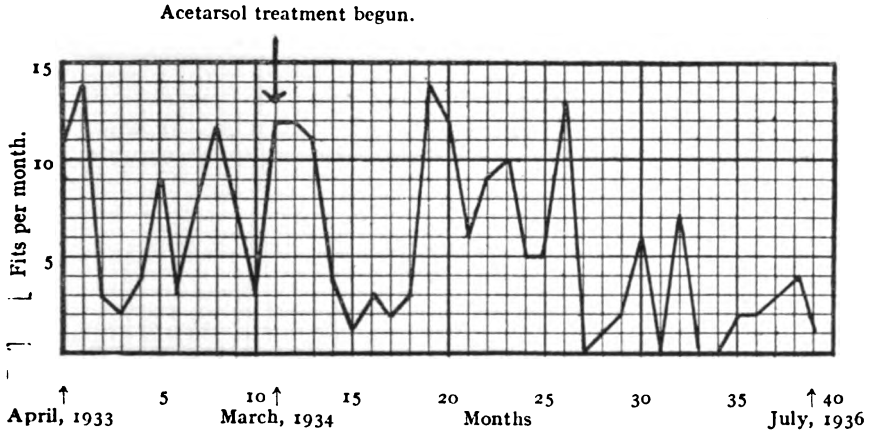
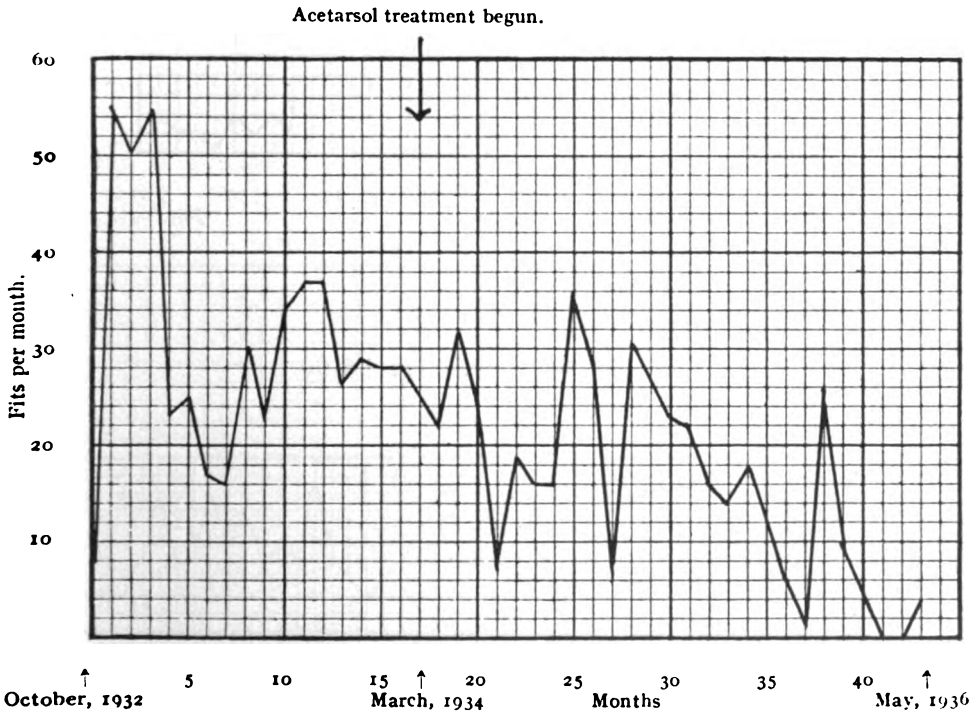
CASE No. 20. Sod. brom. gr. xv twice daily.



CASE No. 4. Pot. brom. gr. xv *t.d.s.*



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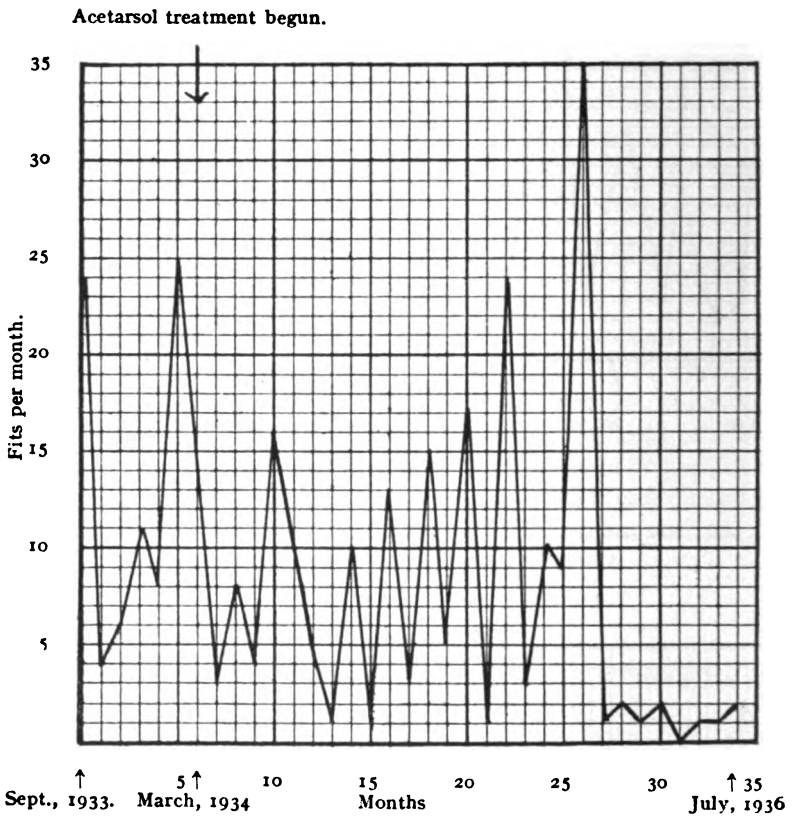
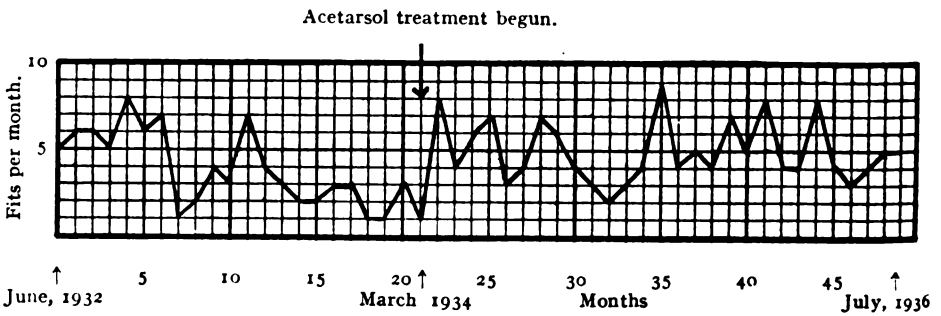
CASE NO. 14. Pot. brom. gr. xv *t.d.s.*



CASE NO. 10. Luminal gr. i daily.



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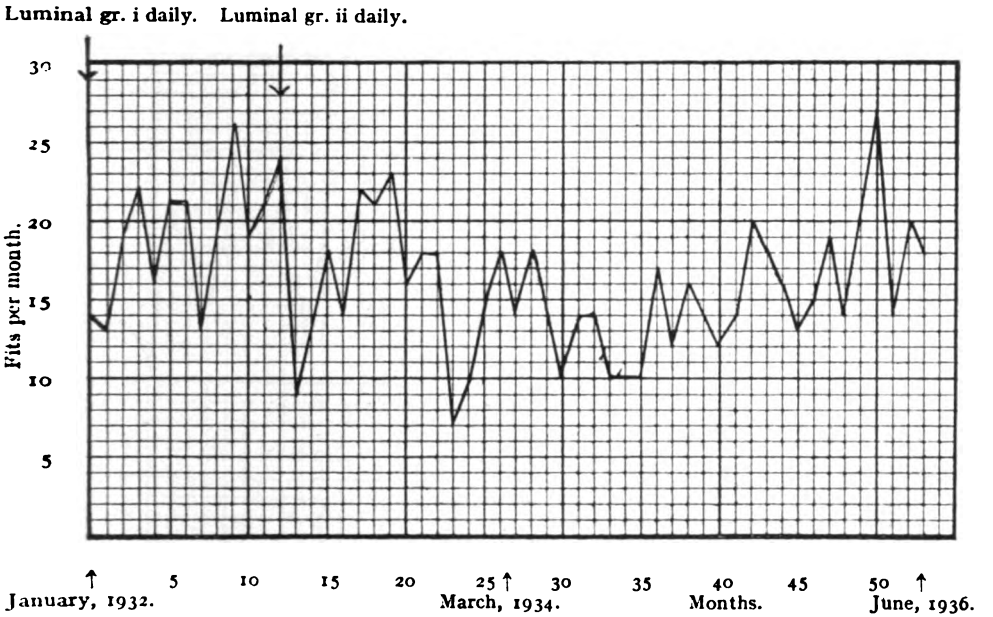
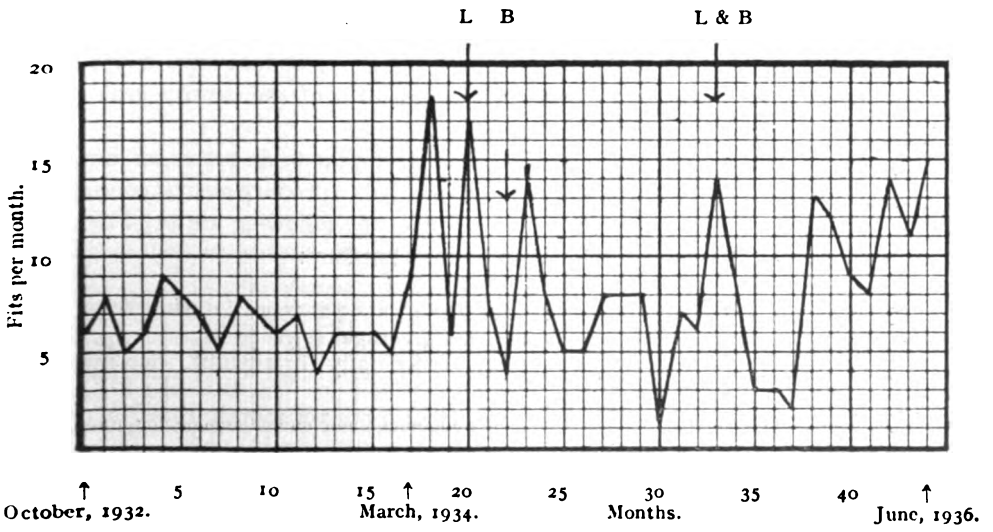
Case. Control No. C4.



Control No. C7.

Treatment: At L, luminal gr. ½; at B, pot. brom. gr. x; at L & B, luminal and bromide in same doses.



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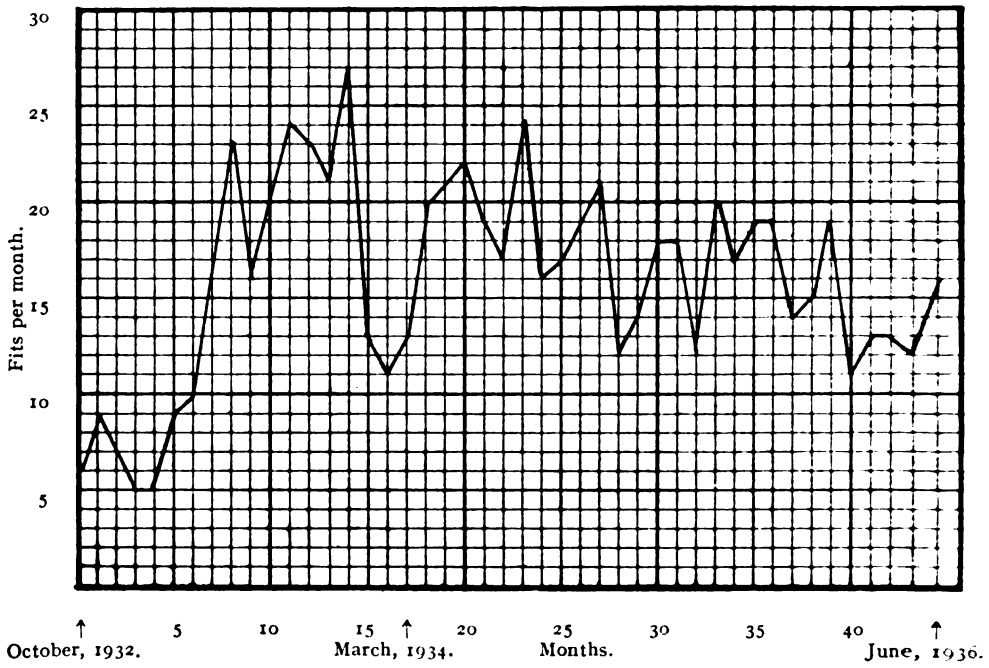
CONTROL No. C9. Luminal gr. i daily.



acetarsol-treated group is mostly, if not entirely due to specific treatment, for the following reasons:

1. In the treatment of epileptics at Caterham (where there are over 650 in residence) the sedative most suitable for the individual case is always chosen. It is pure coincidence that the acetarsol-treated group should have had 4 out of 5 epileptics on bromides continuously, and all 3 of the control group on luminal.

2. On the whole, defective epileptic children appear to do better on luminal than bromides. The latter have often been noticed to produce mental confusion and fits are not always reduced. For example, the following are brief details of two non-syphilitic epileptic boys selected at random:

M. G—, æt. 6 in 1932. Average number of fits per month during 1932 (5), 1933 (3), 1934 (3), 1935 (5), 1936 (7 months' average) (7).

He received pot. brom. gr. xv (grm. 1) twice daily throughout. No mental improvement.

R. D—, æt. 8 in 1931. Had an average of 9 fits per month from March to August, 1931, when he was put on luminal gr. ½ (grm. 0·032) daily. Fits then numbered 14 in September, 20 in October, 8 in November and 3 in December. Dose then doubled. In January, 1932, 2 fits, in February 1, then none until

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November, 1933, when 1 fit was recorded. None during 1934. One in March, 1935, and none in 1936 (7 months). He was much brighter mentally.

In the acetarsol-treated group there were two epileptic children (Cases No. 14 and 20), both on bromides. They have nevertheless done quite well. Fits were reduced in number.

3. Figures are given of average number of fits per case per month in (*a*) acetarsol-treated group, (*b*) control group, (*c*) non-syphilitic epileptic group on bromide, (*d*) similar to (*c*), but on luminal. The acetarsol-treated group is the only one to show any significant reduction of fits.

The non-syphilitic epileptic group was unselected, the only criteria for inclusion being suitability of age and continuous treatment by bromide or luminal over 3½ years.

TABLE V.—*Epileptics.*

AVERAGE NUMBER OF FITS PER CASE PER MONTH.

Groups.	Number of cases.	1933.	1934.	1935.	1936.*
Syphilitic :					
Treated group	5	11	10	9	3†
Control ,,	3	14	13	13	14
Non-syphilitic :					
Bromide ,,	4	7	9	9	7
Luminal ,,	4	7	9	11	9

* 7 months' average.
† Average of 4 cases.

ACETARSOL-TREATED GROUP EPILEPTICS.

Case No.	Age.	Average number of fits per month. 1933.	1934.	1935.	1936.*	Treatment for epilepsy.
4	20	29	23	17	6	Pot. brom. gr. xv, *t.d.s.* throughout.
10	32	3	5	5	5	Luminal gr. i daily throughout.
14	13	11	9	12	1	Pot. brom. gr. xv *t.d.s.* throughout.
17	59	6	3	4 (9 mos.)	Died	Pot. brom. gr. xv *t.d.s.* during August, 1935.
20	11	7	8	5	1	Sod. brom. gr. xv *b.d.*
	Total	56	48	43	13	

* 7 months' average.

CONTROL GROUP EPILEPTICS WITH CONGENITAL SYPHILIS.

Case No.	Age.	Average number of fits per month. 1933.	1934.	1935.	1936.*	Treatment for epilepsy.
C. 4	20	17	13	16	19	Luminal gr. i daily until Jan., 1933; gr. ii after.
C. 7	23	7	9	7	10	Luminal gr. ½ c pot. brom. gr. x varied (*see* graph).
C 9	24	17	18	17	14	Luminal gr. i daily throughout.
	Total	41	40	40	43	

* 7 months' average.

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NON-SYPHILITIC EPILEPTIC GROUP TREATED WITH BROMIDE.

Case.	Age.	Average number of fits per month. 1933.	1934.	1935.	1936.*	Treatment.
D. H—	24	9	14	13	10	Pot. brom. gr. xv, *t.d.s.* throughout.
E. C—	22	9	11	16	12	Sod. brom. gr. xv *t.d.s.* throughout.
M. G—	19	5	4	3	3	Tri. brom. gr. xv *t.d.s.* throughout.
A. B—	26	6	5	3	3	Sod. brom. gr. xv *t.d.s.* throughout.
	Total	29	34	35	28	

* 7 months' average.

NON-SYPHILITIC EPILEPTIC GROUP TREATED WITH LUMINAL.

Case.	Age.	Average number of fits per month. 1933.	1394.	1935.	1936.*	Treatment
E. L—	24	3	2	2	2	Luminal gr. i *b.d.* throughout.
R. D—	27	6	9	15	11	,, ,, ,,
K. H—	25	13	13	16	16	,, ,, ,,
E. S—	22	7	11	10	7	,, ,, ,,
	Total	29	35	43	36	

* 7 months' average.

SEIZURES.

Two of the 3 cases of juvenile general paralysis were subject to seizures.

CASE NO. 24.—The average number of seizures per month was 22 in 1932, 29 in 1933, 26 in 1934, 22 in 1935 and 26 in 1936 (average 7 months). No sedatives given. Treatment by acetarsol appeared to have had little influence on the frequency of seizures in this case.

CASE NO. 21.—This boy had convulsions when aged 1. He had a seizure in June, 1933, and another in November, 1933, then none until June, 1934. Thereafter he had from 1 to 7 seizures per month. For 7 months of 1936 he only had 4, with 3 clear months. No sedatives given. Treatment by acetarsol has been beneficial in this case. Physical and mental improvement coincided with reduction of seizures in 1936.

CLINICAL RESULTS.

The physical and mental condition of the patients were noted before and after treatment. Physical improvement was assessed in terms of general physical fitness, improved muscle tonus, and disappearance of active signs of disease.

Bodily Condition.

Group A: Improvement was marked in 5 cases, good in 11, and moderate in 2.

Group B: Improvement marked in 3 cases, good in 4, moderate in 2, and *nil* in 1.

There was no correspondence between physical and serological improvement.

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Mental Condition.

Mental improvement was judged in terms of general " brightness ", increased interest, better response to training, retentivity and emotional stability.

Many of the treated cases were receiving specialized training by skilled occupational supervisors, and it is not here suggested that the administration of the drug was solely responsible for physical or mental improvement, but only that, in alleviating bodily disease, physical and mental education were thereby facilitated.

GROUP A.—Mental improvement was good in 6 cases, moderate in 10 and *nil* in 2.

GROUP B.—Mental improvement was good in 2, moderate in 5 and *nil* in 3.

GROUP C.—The results in this untreated control group compare unfavourably with the treated groups.

Bodily condition: There was good improvement in 1 case, moderate in 3 and *nil* in 8.

Mental condition: Good improvement in 1, moderate in 1 and *nil* in 10.

As an example of mental improvement in a case from Group A, I quote a report from the occupational supervisor: *

CASE No. 25.—J. E—. Male. Imbecile. Æt. 16. " This boy has definitely improved in school work for some time. His rote memory has improved; he recognizes primary colours but cannot distinguish varying tones of these colours. Auditory powers better but still lacking in fine discrimination. His handwork shows good motor co-ordination as compared with former attempts. General bearing more purposeful, and though still stubborn can give a better account of himself in class work and games."

This boy had, prior to treatment, been attending the school for over 3 years, but little progress had been noted. His cerebro-spinal fluid was abnormal before treatment, became normal after the 2nd course, and remained so throughout (three further tests being done). His blood-serum remains Wassermann and Meinicke positive.

Cases with Active Lesions.

There were two cases of this type. One was an example of active disease with persistent negative Wassermann and Meinicke reactions. Treatment by acetarsol gave very satisfactory results in both cases.

CASE No. 9.—J. S—, female, æt. 33. Family history, 7th of 13 by mother's 1st marriage. Five still-born, 4 died in early infancy, 3 alive, one of whom is stone deaf. By 2nd marriage: 1 miscarriage, 3 still-born, 1 died in early infancy, 1 alive but in poor health and only 1 healthy.

Signs: Peripheral opacities of lens.

* I am indebted to Miss MacAlister for this report.

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On November 1, 1934, she developed a painless ulcer of the pharyngeal wall, about ¼ in. across, with well-defined edges, and dirty whitish base. Treated with gargles for 1 week but no response.

First course acetarsol begun on November 8, 1934. Ten days later, after having received gr. 58 (grm. 3·9), the ulcer was completely healed. This course and 5 others were undergone with benefit.

FIG. 1*a*.—Case No. 19 : Before treatment.

CASE NO. 19.—E. G—, female, æt. 48. Family history, 1st of 12, 5 of whom died in early infancy.

Signs : Advanced double interstitial keratitis. Mitral stenosis.

This patient's blood was Wassermann positive up to 1928, but without treatment became persistently negative. Nevertheless in 1934 she developed a right dacryocystitis, with activation and extension of an old interstitial keratitis of the right cornea. Her hair became coarse, dry, and was falling off freely. Her face was covered by an " acne rosacea ".

Treatment with acetarsol was instituted, and she received 4 full courses,

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at the end of which the facial rash had almost cleared up, her hair was growing profusely and had regained its normal lustre. The dacryocystitis and interstitial keratitis subsided in 2 months. Eyesight was practically *nil*, but it was found possible to perform a high iridectomy, giving a limited vision. Her general health is much improved; she is much brighter and takes much more interest in her surroundings (*see* Figs. 1*a*, 1*b*, before and after treatment).

FIG. 1*b*.—Case No. 19: After treatment with acetarsol.

COMPLICATIONS.

As many as 13 of the 29 treated developed toxic symptoms at one time or another. Only 4 of these 13, however, were finally unable to follow the ordinary dosage according to weight. The chief complications were (1) erythematous skin rashes (6 cases), (2) herpes (3 cases), (3) pharyngitis (3 cases), (4) enlarged cervical glands (2 cases), (5) stomatitis (1 case), (6) aplastic anæmia (1 case, fatal), (7) congestion of lung (1 case), (8) headache (1 case), (9) vomiting (2 cases),

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(10) dysphagia (1 case), (11) drowsiness (2 cases), (12) collapse (1 case). Erythematous eruptions formed the most troublesome of the complications—6 out of 18, or one-third of the females treated were affected. The males, on the other hand, showed singular freedom from skin reactions. Of the 6 females so affected, 3 later developed tolerance and were able to complete treatment on full dosage. The other 3, however, became increasingly intolerant until minimal doses were resorted to.

The type of skin reaction varied from a mild blush to an intense blotchy erythema, rather like that which occasionally follows serum injections. Scarlatiniform or morbilliform rashes were also seen. The usual sites were the arms and legs (outer aspects mostly), neck and trunk. Over the face œdema and diffuse erythema with circumoral pallor were common. Termination was by desquamation of the "branny" or "flaky" variety. Dermal reactions usually make their appearance in from 9 to 42 days from the 1st day of treatment. Once intolerance had appeared, however, this period was considerably reduced. Case No. 12, for example, developed erythema in 6 hours after taking only gr. 1 (grm. 0·06). It was usually sufficient to discontinue administration of the drug for fading to set in. In troublesome cases calcium thiosulphate gr. 9 (grm. 0·6) in 5 c.c. sterile water, intravenously was found effective.

Drowsiness occurred in 2 cases, both epileptics. As one of them had a similar attack a year before treatment, its relation to acetarsol must be doubtful.

Unlike the majority, Case No. 16 did not show any signs of intolerance until the end of the 4th full course; stomatitis with localized dermatitis of the face then appeared. After this the patient could not tolerate more than gr. $\frac{1}{50}$ (grm. 0·0012) three times daily.

Case No. 17 developed an aplastic anæmia at the end of the 5th course. Discontinuance of the drug and the administration of iron with liver extracts had no influence on the course of the disease which went to a fatal termination.

Dermal reactions and eosinophilia.—As previously mentioned all cases showed increase in eosinophil leucocytes after treatment. There was, however, no correspondence between degree of intolerance and eosinophilia. In 4 of the most intolerant cases the highest eosinophil count recorded was as follows:

Case No. 26, 8%.	Case No. 6, 2%.
,, 12, 3·1%.	,, 16, 0·6%.

On the other hand the following cases showed no toxic reactions, and the highest eosinophil count recorded was as follows:

Case No. 1, 23·4%.	Case No. 3, 6·3%.
,, 24, 16·0%.	,, 7, 3·0%.

The development of an eosinophilia cannot therefore be relied upon as an indication of intolerance.

Details of cases developing complications are given below:

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Cases Showing Marked Idiosyncrasy to Acetarsol.

CASE No. 6.—J. C—, female, æt. 19. Family history, little known except that she was the 4th of 5. Had been under supervision from an early age.

Signs: Frontal bossing.

Thirty-seven days from the 1st day of treatment, when she had received a total of acetarsol gr. 501 (grm. 33) and was receiving gr. 6 (grm. 0·39) 3 times daily, she developed a bright patchy erythematous rash of the extremities. Over the palms of the hands it took the form of pale pink spots, slightly raised,

FIG. 2.—Case No. 6: Flaky desquamation of the palms of the hands following treatment with acetarsol.

set in an erythematous background. Face swollen, cervical glands enlarged, tongue dry and coated, pharynx dry and inflamed. No pyrexia. The drug was stopped. Three days later the rash had spread to the neck. An intravenous dose of calcium thiosulphate grm. 0·6 was given in 5 c.c. sterile water. Fading followed soon after. Œdema of the face subsided in two days, and desquamation occurred in five.

Two months later the 2nd course was begun. On the 1st day she received gr. 1½ (grm. 0·096) half an hour before breakfast, the same dose before dinner, and gr. 1¼ (grm. 0·08) half an hour before tea. This was repeated the next day and the day after. One hour after receiving the last dose before tea, she collapsed. Was pale with cyanosed lips and fingers. A few hours later she developed pyrexia up to 102·4° F. Pulse 130. Respirations 40. The next morning an intense scarlatiniform rash appeared over the whole body. Face

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swollen, eyelids puffy, tongue dry and furred, pharynx dry and red, cervical glands enlarged. The drug was discontinued promptly and all symptoms had disappeared in 3 days. Branny desquamation present.

Three days later, acetarsol gr. $\frac{1}{50}$ (grm. 0·0012) *t.d.s.* for 1 week was tried. No toxic symptoms.

The following week the dose was doubled, and then trebled. Finally gr. $\frac{1}{8}$ (grm. 0·01) was given *t.d.s.* for a period of 6 weeks without ill effect.

In the 3rd course the dose was increased up to gr. $\frac{1}{4}$ (grm. 0·15) *t.d.s.* at which level it remained for 6 weeks. Towards the end of the course, however, her face became swollen and eyelids puffy, skin dry. Succeeding courses were, therefore, limited to a maximum dose of gr. $\frac{1}{8}$ (grm. 0·01) *t.d.s.* without any signs of intolerance.

CASE No. 12.—J. T—, female, æt. 14. Family history, not very reliable. Said to be 4th of 6.

Signs: Frontal bossing, saddle nose, rhagades, Hutchinson's teeth, 8th nerve deafness, alopecia, old osteochondritis of wrist, spastic quadriplegia.

Nine days from the commencement of treatment, when she had received a total of acetarsol gr. 22½ (grm. 1·5), she developed a morbilliform rash mostly over the outer aspects of the arms, and over the trunk and buttocks. Pyrexia up to 99° F. Next day temperature normal, rash over buttocks confluent, fading over arms. Four days later rash persisting but not so intense. Calcium thiosulphate grm. 0·6 intravenously in 5 c.c. sterile water, repeated next day and the day after with little effect on the rash. Acetarsol then discontinued. Seven days later the rash had gone, desquamation present.

Two weeks later drug resumed. After receiving gr. 10½ (grm. 0·7) in 1 day she developed an intense erythematous rash over the whole body, accompanied by vomiting and headache. Tongue furred, pharynx dry and inflamed. The drug was discontinued and 2 days later the rash was fading, but gums bled easily, cracks about lips. Five days later rash gone, desquamation present.

One month later acetarsol gr. $\frac{1}{2}$ (grm. 0·32) *t.d.s.* was tried but had to be discontinued 8 hours after the first dose on account of erythema of limbs and trunk. Two months later a similar dose resulted in similar symptoms. After an interval of a week acetarsol gr. $\frac{1}{50}$ (grm. 0·0012) *t.d.s.* was tried, but even this small dose produced an erythematous rash on the 7th day. She was, however, found to tolerate gr. $\frac{1}{50}$ once daily. By giving this dose twice daily at the end of a week and then 3 times daily after another week, it was found possible to treat this patient successfully without further complications.

CASE No. 26.—H. R. C—, female, æt. 12. Family history, 10th of 11. 1st two born dead, next 7 apparently healthy, 11th "backward". Mother's blood W.R. and M.K.R. strongly positive. Father's negative. Patient had "snuffles" after birth.

Signs: No signs of congenital syphilis.

Fifty-five days after the beginning of treatment, when she had received a total of acetarsol gr. 230 (grm. 15·4), she developed a dry, scaly rash about the lips and chin (Fig. 3). Raised erythematous spots over the dorsum of the hands. No constitutional disturbance. Treatment was persisted in, and she received a further gr. 68 (grm. 4·5) in 8 days without aggravation of symptoms, but there was no tendency for the rash to fade. Drug discontinued. One week later rash disappeared, flaky desquamation taking place. Two months later the 2nd course was started, 30 hours after the 1st dose and after receiving only gr. 3 (grm. 0·18) she developed a blotchy erythema over the arms and

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thighs, also over the buttocks. Herpes of lips. No constitutional disturbance. Drug discontinued, rash disappeared in one week. Three weeks later gr. $\frac{1}{4}$ (grm. 0·15) *t.d.s.* was tried, 6 days later similar signs and symptoms developed, disappearing on withdrawal of the drug. One month later gr. $\frac{1}{50}$ (grm. 0·0012) *t.d.s.* was tried, but a week later had to be discontinued on account of early signs of dryness of skin about the lips and chin.

A month later similar symptoms appeared after a week on acetarsol gr. $\frac{1}{25}$ (grm. 0·0024) *t.d.s.* She was then given gr. $\frac{1}{50}$ (grm. 0·0012) once daily for 2 weeks, then the same dose twice daily for the next week, and 3 times daily

FIG. 3.—Case No. 26: Dry scaly rash of face following treatment with acetarsol.

for the succeeding 6 weeks, without any signs of intolerance. All further courses were confined to a dose of gr. $\frac{1}{50}$ (grm. 0·0012) *t.d.s.* throughout, without complications.

Case Showing Late Idiosyncrasy to Acetarsol.

CASE NO. 16.—A. B—, female, æt. 17. Family history, 3rd of 4. 4th said to be dull. Mother deserted family in 1927. Father last heard of in 1928, when he was selling matches.

Signs: Irregular thickening of tibiæ. Irregular scar $\frac{1}{2}$ in. across at lower end of sternum.

Past history: Developed iritis in 1933 and received one course of N.A.B. (8 intravenous injections at weekly intervals, grm. 0·3 to grm. 0·75). After

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the second injection developed pyrexia up to 99° F. with headache and dizziness. Subsequent injections symptomless.

This patient underwent 3 full courses of acetarsol without signs of intolerance. During the 1st course the maximum dose was gr. 6 (grm. 0·39), in the 2nd, 3rd and 4th courses it was gr. 5 (grm. 0·33). At the beginning of the 8th week of the 4th course she developed linear ulceration of the mucous membrane of the mouth corresponding to the cutting margin of the teeth. It started on the left side and spread to the right involving the lower lip. A patch of dry scaly dermatitis also appeared below the right eye. The drug was discontinued.

Total amount of acetarsol received :

1st course	gr. 945	(grm. 63).
2nd ,,	gr. 842	(grm. 56).
3rd ,,	gr. 829	(grm. 54).
4th ,,	gr. 598	(grm. 40).

Two months later the 5th course was begun. At the beginning of the 3rd week, after having received acetarsol gr. 73 (grm. 4·9) she developed ulceration of the mouth and tongue. Drug discontinued for 3 weeks. On resumption, a daily dose of gr. 4½ (grm. 0·3) was given for 5 days at the end of which stomatitis reappeared, with scaly dermatitis below the right eye. Drug discontinued. Two weeks later acetarsol gr. 1 (grm. 0·064) *t.d.s.* was tried, but 3 days later similar symptoms appeared. Drug discontinued. The 6th and 7th courses had to be confined to minimal doses of acetarsol gr. $\frac{1}{80}$ (grm. 0·0012) *t.d.s.* throughout. Any attempt to increase this dose invariably led to the appearance of scaly dermatitis below the right eye.

Withdrawal of the drug always gave relief in from 3 to 5 days.

Cases Showing Moderate Idiosyncrasy to Acetarsol.

CASE No. 7.—M. C—, female, æt. 30. Family history, only child by 1st husband, 4 by second (1st died of tubercle), 3 by third, all apparently healthy.

Signs : Right eye atrophied, left interstitial keratitis with old iridectomy. Scars of old ulcers over body. Absent xiphoid appendix.

Forty-two days from the 1st day of treatment, after having received acetarsol gr. 400 (grm. 26·6) she developed an erythematous rash over the feet and ankles. Treatment was persisted in to the end of the course. Though the rash did not fade until the end of the course there was no tendency to spread.

Six weeks later, the second course was begun. She completed this, as well as 4 others on full dosage, without any signs of intolerance whatever.

CASE No. 13.—D. S—, female, æt. 17. Family history, last of 4. 3rd died 4 months old. Father has been treated for syphilis.

Signs : Frontal bossing, hypoplastic teeth. Choroiditis.

Past history : Patient is reported to have undergone 3 courses of weekly injections during infancy. Suffers from chronic nephritis. On the 34th day of treatment when she was receiving a daily dose of acetarsol gr. 18 (grm. 1·2) and had received a total of gr. 423 (grm. 28·2), she developed an erythematous rash over the arms and trunk. No constitutional disturbance. No increase in albumin-content of urine (0·12%). Drug was discontinued and one intravenous injection of calcium thiosulphate grm. 0·6 in 5 c.c. sterile water given. The rash had completely gone in 8 days' time. In the second

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and subsequent courses no intolerance developed. The maximum daily dose did not, however, exceed gr. 14 (grm. 0·94). The drug appeared to have no ill effects on the already damaged kidneys. Albumin was not increased and few casts only observed. This patient who was often puffy about the face before treatment was much improved physically at the end.

CASE NO. 15.—R. D—, female, æt. 31. Family history, 5th of 7. Nos. 1, 2 and 6 died in early infancy. No. 4, female, æt. 30, married—2 of her 4 children were born dead. Two abortions between Nos. 6 and 7. Father died of a stroke, æt. 40.

Signs: Frontal bossing. Left interstitial keratitis. Pupils unequal, irregular, inactive to light.

Thirty-one days after the beginning of treatment, when she had received a total of acetarsol, gr. 410 (20·3), she developed an erythema of the face, neck and limbs with dry skin. No constitutional disturbance. Next day after taking gr. 12 (grm. 0·8), she vomited. Drug discontinued. Rash gone in 3 days. After 6 weeks' rest she was given a second course in the usual way, but the maximum dose did not exceed gr. 5 (grm. 0·33) *t.d.s.* No further complications developed in this or subsequent courses.

Herpes.

CASE NO. 10.—E. N—, female, æt. 31. Family history, 3rd of 9. 1st born dead, 2nd died, æt. 26. 4th died 7 weeks old. Abortion between 5th and 6th.

Signs: Frontal bossing, scars of old ulcers over trunk. Moderate hydrocephalus.

Nine days after commencement of treatment, when she had received a total of acetarsol gr. 35 (grm. 2·3), she developed a herpetiform rash over the upper lip with pyrexia up to 101° F. Drug not suspended. Temperature normal in 2 days. Herpes away in 5 days. She completed the full course without further incident, as much as gr. 20 (grm. 1·3), being taken daily after the 3rd week. During the 3rd and subsequent courses, however, the maximum dose did not exceed gr. 5 (grm. 0·33) *t.d.s.*, on account of a tendency to "itchy" skin.

CASE NO. 21.—A. E. M—, male, æt. 11. Family history, 1st of 2. Mother has recently been admitted to a mental hospital suffering from advanced general paralysis of the insane.

Signs and symptoms: Parietal regions of skull much thickened.

Rhagades: Central notching of upper central incisors. Eighth nerve deafness. Pupils inactive to light, but react to accommodation, irregular, unequal. Tremors at corners of mouth. Spastic quadriplegia. Persistent grinding of teeth. Seizures. This case is considered one of juvenile general paralysis.

Halfway between the 3rd and 4th courses of acetarsol he developed a herpetiform rash over the left side of the abdomen and extending to the umbilical region, lasting 10 days (Fig. 4). No constitutional disturbance. He underwent 2 further full courses without incident, at the end of which he showed good physical improvement and was much brighter mentally.

Lethargy.

CASE NO. 14.—K. V—, female, æt. 13. Family history, last of 6. 3rd had meningitis in infancy.

Signs: Hypoplastic teeth. Multiple small scars over back. Nodal thickening of tibiæ. Quadriplegia.

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Thirty-seven days after the beginning of treatment, having received a total of acetarsol gr. 391 (grm. 26·1), she developed an acute naso-pharyngitis with enlarged cervical glands. Dysphagia present. No pyrexia, no rash. She was very drowsy at the time. The drug was discontinued and all signs and symptoms had cleared up in 4 days. Ten days later the course was resumed on full dosage without any signs of intolerance. Later four further full courses were taken without incident.

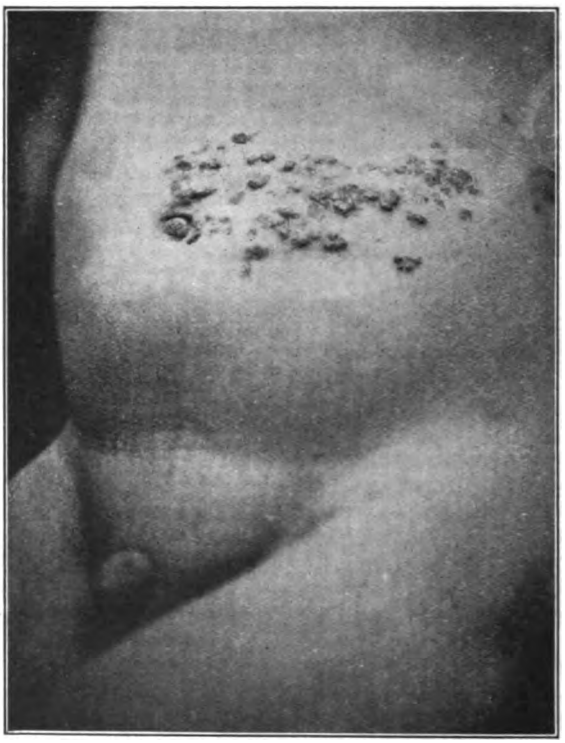

FIG. 4.—Case No. 21 : Herpes of abdomen following treatment with acetarsol.

This patient had suffered from a similar drowsy attack with naso-pharyngitis 6 months before treatment by acetarsol, so that its relation to the drug in the second attack is doubtful.

CASE NO. 20.—R. F. S—, male, æt. 11. Family history, 1st of 4, 2nd is being treated for congenital syphilis elsewhere. 3rd still-born. Mother's blood-serum : W.R. + 30+, M.K.R. ++++.

Signs : Rhagades, hypoplastic teeth.

On the third day of the 1st course, after having received acetarsol gr. 7½ (grm. 0·5), he became drowsy and slowly lost all interest in his surroundings. He was put to bed where he lay apparently asleep for days, though able to be roused to take food. Limbs were semi-flaccid but deep reflexes were present.

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Treatment was continued and he completed the 1st course without showing any mental change. An epiphysitis of the lower end of the left femur developed during this course but had cleared up towards the end. During the interval he brightened up again. In the 6th week of the 2nd course, when he had received acetarsol gr. 238 (grm. 15·9), he again developed similar symptoms of drowsiness. Treatment was this time discontinued and a month later he was sufficiently recovered to complete the course. He received five further full courses without symptoms of drowsiness, but the 5th course was temporarily interrupted on account of the sudden onset of hyperpyrexia of unknown origin.

Pulmonary Complication.

CASE NO. 11.—M. C—, female, æt. 30. Family history, 7th of 13. 6th premature, died. Four others died in infancy. Mother underwent inadequate course of " injections ".

Past history: Patient was a 7½ months' child.

Signs: Rhagades, hypoplastic teeth, right internal strabismus.

During the 5th week of the 1st course, when she had received a total of acetarsol, gr. 462 (grm. 30·8), and was taking gr. 7 (grm. 0·45) *t.d.s.*, she developed signs of pulmonary congestion of the lower lobe of the right lung. The drug was stopped, and 10 days later she was quite well again. The course was then resumed and completed without incident. Six further courses were symptomless, but after the 3rd course the maximum dose did not exceed gr. 5 (grm. 0·33) *t.d.s.* There is some doubt in this case whether treatment was responsible for the onset of pulmonary congestion.

Aplastic Anæmia. A Fatal Termination.

CASE NO. 17.—A. T—, female, æt. 61. Family history, little known. Was one of 16, many of whom had died in infancy.

Past history: Had been in mental hospitals continuously from an early age.

Signs: Scars over palate, osteo-periostitis of tibiæ, dactylitis, onychia, thickening of lower end of radius.

She received 4 full courses without symptoms.

Total amount of acetarsol during 1st course gr. 959 (grm. 64).
" " " " 2nd " gr. 955 (grm. 63·7).
" " " " 3rd " gr. 779 (grm. 52).
" " " " 4th " gr. 812 (grm. 54·2).
" " " " 5th " gr. 797 (grm. 53·2).

The interval between courses was 6 to 8 weeks except between the 3rd and 4th courses, when it was 3 months. During the 9th week of the 5th course it was noticed that the patient was becoming breathless on slight exertion and had a dusky colour. Mucous membranes were pale. The drug was discontinued. Blood examination showed the following: Red blood-cells 3,520,000, colour index 0·8, hæmoglobin 62%. She was given 3 Blaud's pills 4 times daily, together with ½ lb. raw liver daily. Ten days later, on October 19, 1935, red blood-cells 3,300,000, hæmoglobin 8·5 grm. per 100 c.c. (55%), colour index 0·6, white blood-cells 4,800: polymorphonuclears 49%, leucocytes 51%. Red cells irregular in size and shape. No nucleated forms. Van den Bergh reaction negative for both direct and indirect tests. At this time

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she developed a crop of herpes about the nose, and had hæmorrhages from the nose and petechiæ over the palate.

October 10, 1935: Petechial hæmorrhages over chest and forehead. Bruised areas over arms and legs.

FIG. 5.—Case No. 17: Aplastic anæmia following acetarsol. Right femur showing aplasia of bone-marrow with a few hæmorrhagic spots.

October 31, 1935: Becoming worse. Red blood-cells showed marked microcytosis and poikilocytosis. Platelets diminished.

November 2, 1935: 8 c.c. "Hepastab" given intramuscularly. Repeated next day. Half the dose given on successive days.

November 5, 1935: Blood-film stained by brilliant cresyl blue showed reticulocytes and nucleated red cells in fair numbers.

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November 6, 1935: She became suddenly dyspnœic in the afternoon and died soon after.

Post-mortem.—Bones examined, right femur and right ulna. There was almost complete aplasia of the bone-marrow, which was pale yellow with a few scattered hæmorrhagic spots (Fig. 5). All mucosa and internal organs pale. There was a large wedge-shaped hæmorrhagic infarct of the lower lobe of the right lung. The heart was pale, right ventricle dilated, mitral valve thickened. Atheroma of aorta present.

Histological examination of pieces of liver, heart, spleen and kidneys showed no fatty or other degenerative changes.

Cause of death: Pulmonary infarction following aplastic anæmia.

Discussion.

This investigation deals with a mixed group of late congenital syphilitics complicated by mental deficiency, who must be regarded as the least promising for purposes of treatment. None of those treated, however, can in any way be considered "cured". It will take many years of careful observation before a final decision can be given.

Possibly a combination of acetarsol and some other anti-luetic drug, such as bismuth, would have given better results than acetarsol alone, but it would have then been impossible to judge the true value of the drug.

Acetarsol has many advantages over the other arsenobenzene derivatives, the chief of which is ease of administration. Disadvantages such as uncertainty of intake when given to out-patients do not, of course, apply to hospital inmates, especially those of large mental hospitals and mental defective colonies. Dermal reactions are certainly troublesome; many, however, develop subsequent tolerance and can undergo full dosage. A few prove quite intolerant, and can only take small doses. In support of minute dosage in such cases, I will quote no less an authority than Hutchinson, when mercury was the drug of choice: "In the employment of specifics in the treatment of syphilis, everything may be said to depend on dose. The statement so often made respecting patients, that they 'cannot bear the smallest quantity of mercury or iodide', may always be set down as a mistake. Reduce the dose sufficiently and the drug will be borne, and when it is borne it will cure. It is not quantity of the drug which is needed, but its effect on the organism, and if the specific effect is gained by a minute quantity it is not only not needful, but bad practice to attempt to increase it. I have never yet met with a patient who could not take either iodide of potassium or mercury, if only the dose was sufficiently reduced. The chief difficulties in treatment occur in those who are insusceptible, not in those who respond easily. I have repeatedly reduced the iodide of potassium and grey powder, respectively, to one-sixth of a grain for a dose, and found them to agree well and to manifest specific influence."

The 3 cases showing most intolerance to acetarsol and who were treated with small doses improved both physically and mentally. In one case the

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blood Wassermann reaction was changed from positive to negative and the M.K.R. reduced in strength. In the second the Wassermann reaction was reduced in strength and the M.K.R. unchanged. In the third case the Wassermann reaction of the cerebro-spinal fluid was changed from strong positive to weak positive and the increased cells and protein fell to within normal limits. Lange's colloidal gold curve, originally strongly paretic, became weakly paretic, the blood Wassermann reaction and M.K.R. remained positive —this case only received gr. $\frac{1}{50}$ of acetarsol three times daily throughout. On the other hand another patient on full dosage showed no serological change after treatment—does he belong to the "insusceptible" class of Hutchinson?

As a result of experience the following scheme of dosage is recommended:

1. Those showing no idiosyncrasy, dosage and duration of course according to that advised by Bratusch-Marrain. Maximum daily dose not to exceed gr. 15 (1 grm.).

2. Those showing moderate idiosyncrasy, drug discontinued promptly on appearance of first symptoms. Two weeks' rest after the disappearance of symptoms, drug then resumed, gr. $\frac{1}{12}$ (grm. 0·005) three times daily for 1st week, gr. $\frac{1}{8}$ (grm. 0·01) three times daily for the second, and so on, doubling the dose at the end of each week until either the normal dosage according to weight is attained or symptoms of intolerance develop. In the latter event the case is placed in the category of extreme idiosyncrasy and is treated accordingly.

3. Those showing extreme idiosyncrasy, drug discontinued promptly on appearance of first symptoms. If reaction severe and of dermal type, calcium thiosulphate grm. 0·6 in 5 c.c. sterile water given intravenously. Two weeks rest after disappearance of all symptoms. Drug then resumed, gr. $\frac{1}{50}$ (grm. 0·0012) daily, as a single dose before principal meal, for one week. Increased to twice daily during second week, and then three times daily for the third week. The dose may then be doubled in the fourth and, failing symptoms of intolerance, trebled in the fifth week. Usually mild signs of intolerance will appear before this, in which case the drug is stopped for one week, and is then resumed at a dose just below that which produced intolerance, and maintained at this level throughout. Each complete course lasts 9 weeks, with similar intervals to those taking full dosage.

For all three classes the minimum number of courses should be six, irrespective of any change in serological reaction.

Results of Treatment.

In the group of cases with normal cerebro-spinal fluids, of 17 with positive Wassermann reactions 8 became negative at the end of just over two years. In Davidson and Birt's series 14 out of 23—with presumably normal cerebro-spinal fluids—became negative in 2 years. The better results obtained by

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these authors are probably due to the average age of their group being much lower. Even in the Caterham series there is a tendency for the younger members to become serologically negative more easily than the older ones.

The serological reactions of the blood sera of cases with abnormal cerebro-spinal fluid appear to be extremely resistant to treatment, only one of nine becoming negative. The treatment of congenital neuro-syphilis in older children is unfortunately very disappointing. Nabarro is of opinion that there are only two forms of neuro-syphilis worth treating: (1) that which occurs in early infancy and (2) latent neuro-syphilis of older children. "Clinical neuro-syphilis, although it may be considerably benefited by treatment, does not, in my experience, result in the production of cure or of an individual who is going to be an asset to the community." Again, "and nowadays I regard the treatment of congenital neuro-syphilis with symptoms as practically hopeless."*

Although acetarsol has been shown not to penetrate into the cerebro-spinal fluid, it nevertheless appears to exert a specific effect on the majority of abnormal fluids. Pleocytosis is the first abnormality to disappear, next increase in protein, and then Lange's colloidal gold curve; lastly the Wassermann reaction, though reduction in strength is fairly common. Even in cases with definite juvenile general paralysis the cerebro-spinal fluid has tended to improve. A reduction in the frequency of fits in congenital syphilitic epileptics is perhaps to be expected with any form of effective treatment, so it is not surprising to find a reduction of fits in 7 of 8 such cases. Diminution in the number of seizures in one case of juvenile general paralysis was, however, unexpected; it coincided with mental and bodily improvement and was considered a "remission". The majority of those treated improved both physically and mentally to some degree, weight was gained and hæmoglobin and red-blood corpuscles increased in a number of cases. Such all round improvement is fairly common in congenital syphilitics treated with other forms of arsenic and is not confined to acetarsol.

Two cases of congenital syphilis with active signs responded to treatment with acetarsol as well as might have been expected with N.A.B. or other similar preparation.

Complications.

Bratusch-Marrain states that where an overdose of spirocid is given diarrhœa may occur. Semon, in 1923, was of opinion that stovarsol's relative toxicity as compared with the trivalent N.A.B. was great, as a single 4 gr. (grm. 0·27) tablet was said to contain more arsenic than an ampoule of 0·3 grm. of neo-salvarsan. He described a lady, æt. 42, who developed a rash resembling German measles on the 9th day of treatment. On the 14th day

* Communication from Dr. Nabarro to Dr. Lindsay, quoted by their kind permission.

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there was swelling of hands and feet, œdema of lids and lips and conjunctivitis. Petechiæ on fingers and feet, thighs and buttocks. Cervical glands enlarged. Nausea complained of, but no vomiting or diarrhœa. As only 40 gr. (grm. 2·67) had been administered in 13 days he suggested an inherent susceptibility to the drug.

In 1925 Izar mentioned 4 cases in which moderate doses of stovarsol were followed by severe symptoms of vertigo, collapse, cyanosis, abdominal pain, pyrexia and tachycardia. He attributed the results in these cases to individual idiosyncrasy. In 1926 O'Brien reported a case of a man, æt. 46, who, after the ingestion of a single 4 gr. (grm. 0·27) tablet of stovarsol one hour after breakfast developed within two hours severe cramp-like pains over the abdomen and lower limbs. There was much vomiting for two hours and also diarrhœa, prostration and collapse. He suggested that stovarsol may produce these symptoms after food.

In 1927 Bender reported 6 cases of poisoning by stovarsol. He stated that " mention of undesirable effects is present in nearly every report of its use in a series of 10 or more cases, including one or several of the following manifestations—coryza, bronchitis, sore throat, malaise, aching, chill, skin eruptions, jaundice, adenitis, diarrhœa, abdominal pain, eosinophilia and albuminuria ".

In 1929 Michael reported a case of exfoliative dermatitis in a woman. Two tablets of acetarsone each 0·25 grm. were given at 10 a.m. and 2 p.m. Eight hours after the second dose she experienced burning and itching of the face and arms ; these parts became congested and exudative, eyelids were swollen, and there was moderate abdominal cramping and diarrhœa.

Recently Davidson and Birt stated that only 3 patients out of 51 treated developed mild toxic symptoms, 2 had headache relieved by stopping the drug, and not reappearing when resumed, the third had coryza, gastro-intestinal distress and diarrhœa. They state " there have been fewer toxic effects in this series than is usually found with arsphenamine or neoarsphenamine. Those that have been produced were readily controlled by dosage ".

In the Caterham series, 13 out of 29 cases developed toxic symptoms of one kind or other, dermal reactions being the most common ; other manifestations were fever, herpes, pharyngitis, adenitis, stomatitis, congestion of the lungs, headache, vomiting, dysphagia, drowsiness and collapse. Aplastic anæmia occurred in one case. Contrary to most observers' findings, diarrhœa did not occur. Drowsiness for long periods appears rather unusual, and has not been previously reported. I have not been able to trace any report of the development of aplastic anæmia following stovarsol. Findlay states that " just as benzene poisoning is known to cause an aplastic anæmia so the arsphenamines may also, in rare cases, produce an aplastic anæmia with degeneration of the bone-marrow ". It will be noted that this fatal case of aplastic anæmia occurred in a woman whose age was 61. She was originally included in the series so as to make the group as representative as possible of all ages. It is,

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however, doubtful whether much good can result from the treatment of a latent congenital syphilitic of her age merely because she had a positive Wassermann reaction and M.K.R. of the blood. It is suggested that similar cases should, if necessary, be treated by less drastic methods.

Summary.

1. Twenty-nine mental defectives, the subjects of congenital syphilis, were treated with acetarsol. The majority underwent 6 to 7 courses of 9 weeks each, over a period of just over 2 years.

2. In one case it was shown by direct experiment that after ingestion the concentration of the drug in the systemic blood-stream was still rising after 24 hours. The drug failed to penetrate into the cerebro-spinal fluid.

3. The scheme of dosage was based on that of Dr. Alois Bratusch-Marrain with modifications, details of which are given.

4. For analytical purposes the 29 cases were divided into two main groups: (*a*) those with normal cerebro-spinal fluids, and (*b*) those with abnormal cerebro-spinal fluids.

Group (*a*).—19 cases, 17 of whom had positive blood Wassermann reactions before treatment. After treatment 8 became negative, 7 reduced in strength, and 2 unchanged.

Group (*b*).—10 cases, 9 of whom had positive blood Wassermann reactions before treatment. After treatment, one became negative, one slightly reduced in strength, and 7 unchanged.

The M.K.R. blood reactions of both groups, though more resistant, showed similar trends.

5. Of 10 abnormal cerebro-spinal fluids before treatment, 3 became completely normal and 7 were improved after treatment.

6. A control group of 12 untreated congenital syphilitics with normal cerebro-spinal fluids was observed over approximately the same time as the treated group. The following serological changes took place. Of 7 positive Wassermann reactions 1 became negative, 4 were unchanged, and 2 increased in strength. One doubtful and 4 negative Wassermann reactions became positive.

7. Blood analyses were done in 25 cases. Thirteen showed increase in hæmoglobin, 21 increase in red blood-corpuscles. All showed increase in eosinophil leucocytes.

8. Twenty-seven cases improved bodily and 23 mentally. Twenty-two gained weight.

9. Of 8 epileptics 7 showed reduction of fits.

One of 2 cases of juvenile general paralysis showed reduction in number of seizures.

10. Two cases had active lesions before treatment. One with a pharyngeal

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ulcer, healing took place in 10 days. The other, with dacryocystitis and interstitial keratitis was well in 2 months.

11. Toxic manifestations occurred in 13 cases; only 4 of these, however, were unable to follow the usual dosage according to weight and were treated with modified courses. Dermal reactions were the most frequent and troublesome of the complications. One case, æt. 61, died of aplastic anæmia, with sudden onset at the end of the 5th course.

Conclusion.

Acetarsol is a drug with a powerful salvarsan-like action on cases of congenital syphilis. It has the added advantage of oral administration, rendering possible the treatment of large numbers of cases with the minimum of apparatus and great saving in time. It will be found very useful in those large mental hospitals and mental defective colonies where patients are under constant care and supervision. If the scheme of dosage as given in this paper is adhered to there will be little likelihood of serious toxic reactions resulting. The close relationship of acetarsol to tryparsamide, and the fact that many abnormal cerebro-spinal fluids were improved and 2 cases of juvenile general paralysis benefited, suggests the desirability of further research along these lines.

Perhaps treatment aided by other antisyphilitic drugs may be found more effective, but there is little doubt that acetarsol is entitled to a place in the modern treatment of congenital syphilis.

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ACROCYANOSIS.*

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INTRODUCTION.

WHEN normal human skin is moderately cooled it goes blue, owing to a contraction of the arteries and arterioles, and a relaxation of the capillaries and subpapillary venous plexus of the epidermis. This blueness is light in colour. In certain individuals, however, it is deep, or relatively saturated, in the hands or feet or both; these include cases of acrocyanosis (ἄκρον = extremity, κυάνεος = dark blue). This condition, though rare in the general population, is common among the inmates of mental institutions.

The latter fact has long been familiar to alienists, and is mentioned in the standard text-books. Stoddart (36) states that in stupor the extremities are often cold and blue, and Henderson and Gillespie (12) note that in schizophrenia the hands, feet, nose and ears (*sic*) show cyanosis and œdema. Similar conditions are found in mental defectives, Tredgold stating (37) that in "mongols" cyanosis and coldness of the extremities are prevalent.

Various hypotheses have been proposed as to its ætiology, course and pathology, but none has seemed satisfactory. It was therefore decided to re-investigate the condition.

It was found that the existing clinical descriptions were incomplete and that nothing further was certainly known. The reasons for previous shortcomings seem to lie in the study of insufficient numbers of cases observed for an insufficient length of time, and a failure to appreciate the mechanics of the small blood-vessels or to make histological examinations.

In this investigation some hundreds of cases of the disorder have been examined, and, in many of these, careful observations have been made over a period of years. Theoretical considerations early led to the prediction of certain morbid histological changes and this was confirmed. A special examination of the blood has also been made in six cases. All the results have been consistent with the same conclusions.

It will be shown that acrocyanosis is due to certain changes in the arterioles caused by continual cooling of the parts, and that the condition is not always permanent. In order to detect it in all cases, special methods have had to

* A dissertation for which the Bronze Medal of the Royal Medico-Psychological Association was awarded, 1937.

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be used, revealing that it is more prevalent than has been thought. It is approximately symmetrical on the two sides of the body.

History and Clinical Features.

Crocq (7) first used the word "acrocyanosis" to describe a condition in which the hands and feet had slowly become permanently blue, the discoloration ending gradually at the wrist and dorsum of the foot respectively. The colour was deeper on the volar surfaces, which were moist, than on the dorsa, which were dry and cold; it was accentuated towards the toes. Local pressure caused a white spot, which disappeared slowly. There was no œdema, ulceration, paresis, severe pain or contracture. It was readily distinguished from the local syncope of Raynaud's disease, and from Charcot's "blue œdema" with its paresis, contractures and severe pain. Crocq described two cases of acrocyanosis, both hysterics. One of them also suffered from arthritis followed by pleurisy, the other recovered from acrocyanosis when her hysteria was cured.

In two particulars Crocq's description needs amplification. As will be shown, the cyanosis is not permanent, except perhaps in the most severe cases, but it will appear to be so if the patients are examined only when the parts are cool. Secondly, œdema is constantly absent only from the milder cases.

The colour of the cyanosed skin is not uniform, and it usually contains bright red areas—so-called cinnabar red spots (10, 13); it is generally between X and XV on Lewis's scale (23), the colour approaching full cyanosis; above all it is exceptionally deep. The depth of colour depends partly on the position of the parts, being greater when they are dependent. The white spot which can be caused by local pressure, and was noted by Crocq, disappears spontaneously in a very characteristic way, the colour returning from the periphery only, and not also from below as in normal skin. Local œdema is fairly often present; it may merely cause the skin to feel a little tight—a condition seen typically in the fingers—or give rise to visible swelling, in which case pressure causes the usual pitting—a fact which has been denied (8, 18, 38). Chilblains are perhaps a little commoner than normal, but in their absence ulceration and gangrene do not occur, although in severe cases the nails become irregular and brittle, and in the most extreme examples examined (in mental defectives) the skin becomes dry and scaly. The syndrome appears to cause remarkably little disability, and mental patients do not complain of any subjective symptoms, but lesions of the skin in the affected areas readily become septic and heal slowly. In hot weather, and when the subjects are warm or active, the hands and feet may be distinguished from the normal by their clamminess, by a brownish-yellow tinge on the backs of the hands, or in some cases not at all.

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This brownish-yellow colour, which has not previously been reported, ends just above the wrists and is most obvious in the summer. It is presumably due to the fact that since the hands are kept still and exposed, they become more tanned by the sun than those of normal subjects. It was only seen on the feet of one patient (A. P—, Appendix, Case 6), and she had not been wearing stockings.

Clamminess of the palms and soles was noted by Crocq and others since him (18, 38), but no further details were given. In this investigation numerous observations with a hand lens have shown that, when present, it was invariably due to liquid issuing from the sweat-ducts. Now Kuno (16) has shown that perspiration at low temperatures occurs more on the palms and soles than elsewhere, is increased by hyperæmia, and, failing to evaporate, collects on the surface. These are precisely the conditions we have in acrocyanosis. If, however, the circulation to the skin ceases, perspiration will fall to a minimum, and this explains the dryness of the parts in Raynaud's phenomenon. He thinks that the greater sweating on the palms and soles serves to keep the thick stratum corneum moist and pliable. It seems probable that in cases who stand about unoccupied this layer atrophies, and the perspiration, being more than sufficient, accumulates and makes the skin wet. The opposite extreme is familiar in the dry, horny palm of the labourer. It is, of course, conceivable that excessive sweating of the parts precedes the development of acrocyanosis—a possibility that will be discussed below (p. 414).

There was no evidence of severe sensory changes in this series. The mental condition of the cases investigated was such that subjective sensory observations could not be made.

Differential Diagnosis.

Acrocyanosis is frequently complicated by erythrocyanosis crurum puellaris, cutis marmorata, which may be extensive, and the pernio follicularis of Klingmüller and Dittrich (13). There may thus be a considerable amount of cyanosis in other parts, and the condition of the patient may bear a superficial resemblance to general cyanosis from a central cause; the pattern and distribution of the skin colour is, however, quite distinctive, and red areas can always be produced by local rubbing or other trauma. The complicating conditions mentioned at the head of this paragraph are themselves easily distinguished from acrocyanosis, as none of them affects the hands or feet.

Thrombo-angiitis obliterans is differentiated by its progressive character, the frequent presence of so-called trophic lesions, and the absence of pulsation in the arteries of the part.

The chief disorder likely to be confused with acrocyanosis is Raynaud's disease. But the former does not cause ulceration or gangrene, hence the small depressed scars, so often seen on the tips of the fingers in cases of

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Raynaud's disease, are absent. Local trauma, such as pricking, rubbing or pinching a small area, causes local redness within the surrounding blue of acrocyanosis; but in Raynaud's disease restoration of colour can only occur by an orderly spread from the proximal end, so that at a given instant all parts distal to a cyanosed area must themselves be cyanosed, and the cinnabar red spots, so characteristic of acrocyanosis, are not seen.

The accurate use of the term "acrocyanosis" has not always been observed, e.g., Barker and Sladen (1) have described under this name cases which showed ulceration and gangrene, and appear more probably to have been examples of thrombo-angiitis obliterans. It is indeed uncertain that all the cases collected here from the literature are of the same type, originally described by Crocq, as those investigated below, but the similarity seems such as to justify that conclusion.

Induction of Attacks.

It has been found in this investigation that, if patients known to suffer from acrocyanosis are examined out of doors on an average winter's day, a proportion of them only will manifest the condition. On another such occasion it will be noticed that some of those previously affected are active and warm, with pale and not blue extremities, whilst others, who previously appeared normal, are now seen to be acrocyanotic. This is because the cyanosis of the extremities is partly dependent on their temperature, being most severe with a moderate degree of cold and gradually disappearing as the temperature rises. It explains the observation of Claude and Baruk (7) and Layani (18) that the degree of acrocyanosis varies with the amount of catatonia, for when the latter decreases the patient will be more active and therefore warmer. It also explains Crocq's reported cure of his second case (7), for she probably kept her hands warmer after mental recovery. Patients who do not show this syndrome merely because at the time of examination their extremities are warm may be suitably called cases of latent acrocyanosis—a description which will be used below. In such cases it will be necessary to cool the hands and feet in order to demonstrate the syndrome.

Cooling to the region of 0–10° C. is of no avail, for it makes the hands red. This redness was found by T. Lewis (27) to interfere with the induction of attacks of Raynaud's phenomenon, and it prevents the occurrence of attacks of acrocyanosis for reasons which he gives: dissociation of oxyhæmoglobin is slow at these temperatures, and excessive cold causes dilatation of the arterioles —a reaction discussed below.

Reactions of Normal Skin Vessels to Ice and Iced Water.

T. Lewis (26) showed that local hyperæmia occurred when the normal skin was cooled to the region 0°–10° C. It was believed that the H-substance (22) was liberated and gave rise to an axon reflex, but though local redness and

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rise of temperature occurred, and there was a diffuse erythema around the cooled area, this could not be shown to be of reflex origin, since it might equally well have been caused by conduction of cold along the skin from the cooling agent.

If, however, the absence of such erythema around the cooled area could be demonstrated in cases in which the cutaneous sensory nerves had degenerated, it would be evident that the erythema noted above was due to an axon reflex. This was shown by the following observations :

(I) Mrs. H— was first seen in May, 1930, when she had a small abscess, midway down the inner side of the left leg, opened by a transverse incision. The abscess subsequently healed, but she showed sensory changes corresponding to division of the long saphenous nerve at that point.

On July 9, 1930, there was an area of anæsthesia to cotton-wool over the whole of the anterior, posterior and medial surfaces of the lower third of the left leg, and of analgesia to pin-prick 6·5 cm. broad on the medial surface of this area, and here a scratch did not cause any surrounding " flare ". Sensation in the right leg was normal.

Both legs were immersed in water at 30° C. for ¼ hour, and were then withdrawn and gently dried ; their temperatures, measured with a thermal junction, were found to be equal. A lead capsule, through which ice-cold water circulated, was then applied to each leg in turn. The temperature of the outflow was found to vary between 2·7° and 4·5° C. On the right leg a flare was seen extending for a total distance of 4·5 cm. from the edge of the capsule ; on the left leg no flare was seen in the analgesic area.

(II) A similar observation, using a small block of melting ice as the cooling agent, was made on December 31, 1931, on a patient, J. Y—, who had had the left tenth thoracic intercostal nerve blocked with alcohol on November 14 previous, for relief from the pain of an attack of herpes zoster.

Thus the two elements, local redness and flare, of T. Lewis's " triple response " (22) are seen when the normal skin is cooled to the neighbourhood of 0–10° C. The third element, œdema, is observed when the skin is more strongly cooled, as was found by T. Lewis and Love (29). These observations support the view that the H-substance is liberated by cooling of the human skin below 10° C.

It will now be evident that strong cooling of the skin, far from inducing attacks of acrocyanosis in latent cases, will, by releasing the vasodilator H-substance, actually prevent their development. A striking confirmation of this is that on exceptionally cold days, subjects of this malady are found to have bright red, and not blue, hands.

Ætiology.

Numerous suggestions have been made, but the logic of many of them is exceedingly difficult to follow. As is usual in obscure conditions, almost cryptic causes have been involved.

Delater and Hugel (8) described acrocyanosis amongst various other

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erythrocyanotic changes in the limbs, all of which they consider to be due to an endocrine-neuro-vegetative disorder. Layani (18), who has written a monograph on this syndrome, thinks it is due to pluriglandular endocrine dysfunction acting through an abnormally labile autonomic system. Villaret (38) believes the cause to be endocrine, especially ovarian, except in such cases as catatonia, Parkinsonianism and hemiplegia. Kreis (15) does not consider it to be a true clinical entity, but to be due to a taint with hereditary syphilis. He states that, though there is probably a connection with the female reproductive organs, this will not allow us to seek the origin in an ovarian deficiency, since "Ce recours est équivalent à l'explication d'un phénomène inconnu par une nouvelle inconnue".

Most other authors, however, favour exposure to cold as the cause. Heidenfeld (11) and Moeller (32) have described cases of the allied condition erythrocyanosis crurum, which they believe to be due to the insufficient warmth in the modern clothing of young women. Klingmüller and Dittrich (13) include various cutaneous cyanotic conditions in the term "perniosis", and consider them all to be consequences of cold and fall in external temperature. Haxthausen (10) follows these two workers, and points out that the situation of these maladies on the face and hands, and in females the lower limbs, is consonant with such a cause, and also that the lesions have a certain similarity to the acute effects of cold. Nevertheless, he thinks there is another factor also concerned, and that acrocyanosis in particular "is the expression of a more diffuse, but not very intensive action of cold upon the skin, or rather that it is that form of perniosis in which the effect upon the vessels of the predisposing factors is particularly evident in comparison with that of the exciting factor—the cold".

In the course of these investigations it has been found that acrocyanosis occurs with striking frequency in catatonic dementia præcox. The observation is not a new one. Layani (18, 19) has called acrocyanosis in catatonics "cyanose de décliveté',' which is perhaps best translated as "gravitational cyanosis". Villaret (38) includes it among his "symptomatic acrocyanoses".

It was decided to investigate acrocyanosis in the patients occupying two subacute-chronic wards, one male and one female, in the Napsbury Mental Hospital of the Middlesex County Council. They were selected as a representative sample of mental hospital population in this country. Fifty-five patients of each sex were investigated. They wore ordinary loosely fitting clothes, with boots when out of doors, woollen hose, and no gloves. The observations on the women took from June to August, 1931, and on the men, from the latter month to January, 1932. In each case if an attack was not in progress, the hands and feet were cooled by immersion in water at 20° C. until an attack occurred or for ½ hour. Actually only a small number of those affected were seen in a spontaneous attack, and in the absence of artificial cooling to a suitable temperature they would have escaped notice altogether.

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The cases were classified by their psychiatric diagnoses, by whether they habitually stood about or were normally active, and by the degree of acrocyanosis obtained.

Diagnosis.	Habits.	No acrocyanosis.	Mild acrocyanosis.	Marked acrocyanosis.
Dementia præcox	Still, 45	14	5	26
	Active, 22	15	1	6
Mania	Still, 0	..	..	..
	Active, 8	6	1	1
Melancholia	Still, 6	3	1	2
	Active, 2	2	..	..
Delusional	Still, 3	2	1	..
	Active, 11	8	2	1
Epileptic	Still, 0	..	..	..
	Active, 7	5	1	1
Mental defect	Still, 2	1	..	1
	Active, 4	4	..	..
Totals	Still, 56 (100%)	20 (36%)	7 (12%)	29 (52%)
	Active, 54 (100%)	40 (75%)	5 (9%)	9 (16%)

There were 55 cases of each sex; altogether 26 men and 24 women had acrocyanosis.

It will be seen from the table that acrocyanosis is two-and-a-half times as common in those who habitually stand or sit about and allow themselves to be chilled as in the normally active. No other factor was found to bear any constant relation to it, although in each case the blood-pressure, full mental and physical state and, frequently, the blood Wassermann reaction were examined. There was no evidence of endocrine dysfunction.

In order to obtain still more reliable evidence, 49 of these patients were re-examined at the end of nearly four years; the results point to the same conclusion. It was found that 13 (26%) showed decreased activity, leading to greater cooling of the extremities, and in all of these the degree of acrocyanosis was found to be increased. Six (12%) showed the contrary changes of increased activity and warmth with decrease of acrocyanosis. Two (4%) inactive cases had developed acrocyanosis without change in their habits. The remaining 28 (58%) showed no change in either way. These 49 cases were selected only because they were the most readily available, and it is striking that the results, without exception, indicate continued cooling of the parts as the cause of acrocyanosis.

It is possible that, in some cases, cooling may arise indirectly from mental stress and so cause acrocyanosis, for Kuno has shown (16) that sweating on the palms and soles is principally caused, not by heat, but by emotion. This will, of course, cool the part. It has been demonstrated above that acrocyanosis results from continued local cooling. In the following case emotion may thus indirectly have caused a mild acrocyanosis:

J. H—, a schoolgirl, aged 13½, was referred to the North-Western Child Guidance Clinic in March, 1935, on account of emotional difficulties. She was precociously developed, but presented no other physical abnormalities.

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She stated that she usually suffered from broken chilblains on the fingers and toes in the winter, and that for about three years her hands had been noticed to go unduly blue when cold. Her mother confirmed this.

When examined on April 18, 1935, at a room temperature of 17° C. her hands were slightly bluish and warm except for the fingers. The palms were moist, and beads of sweat were seen at the orifices of some of the ducts on the fingers. After 15 minutes' immersion in water at 20° C. the right hand was colour XII–XIII, the left XIV on Lewis's scale (22), i.e., they were cyanosed. Both were pale, could be paled further temporarily by elevation or light pressure, and trauma caused local reddening.

The circulation in the feet was found to be normal. The blood-pressure was 107/71.

Incidence.

Most of the cases of acrocyanosis in this investigation were between 20 and 45 years of age. This is a little below the average age in mental hospitals, probably because as age advances catatonia decreases and the patients keep warmer. The youngest was an epileptic idiot aged 4 years and 9 months (a fairly severe acrocyanotic), and the oldest a paraphrenic aged 67 (a mild case of acrocyanosis), but it is not suggested that these represent limits.

Layani (18) and the French authors state that acrocyanosis is commoner among females. The table given above (p. 414) does not support this contention, for of 110 comparable cases consisting of 55 of each sex, 26 men and 24 women were affected. It may be, however, that in non-psychiatric practice women are more likely to seek advice for a condition that is unsightly rather than disabling.

It will be noticed from the same table that the syndrome is not confined to any type of mental disease, although it is particularly common in dementia præcox and rare in mania. It is apparently rather exceptional in the general population, though known in dermatological practice. Nevertheless it is important, for 50 patients out of 110, chosen as an average sample of mental hospital population, showed demonstrable acrocyanosis, only 18 of them being mild cases. Therefore, since the number of persons suffering from mental disorder, notified as under care in England and Wales on January 1, 1934, was over 150,000 (2), the condition must affect some thousands.

Mechanism.

It is customary to dismiss the question of mechanism of acrocyanosis, chilblains and the like, by attributing them to a "poor circulation". This vague phrase, which has been a veil for much ignorance, is best avoided, "poor peripheral circulation" being preferable if some such expression be necessary.

Boas (3) found the capillary blood-pressure to be lowered in acrocyanosis, and the capillaries thickened with a slow stream, which was accelerated in hot water. Delater and Hugel (8) state that there is spasm or endothelial

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proliferation of the last arterial branches in acrocyanosis, but they do not give their evidence. In the allied condition of perniosis they found distension of the capillaries and of the veins of the subpapillary plexus, and also peristaltic inco-ordination, which they consider to be the primary factor involved. Nielsen (33) reports stagnation of the blood in the minute vessels in erythrocyanosis crurum, as also does Dittrich (9) in the various conditions he includes in the group of perniosis. The last author describes various histological changes in the vessels of the deeper layers, including thickening of the muscular and intimal coats, and thrombosis. His work is of a painstaking nature, but suffers from the confusion consequent on describing such obviously different conditions as acrocyanosis, erythrocyanosis crurum puellaris and chilblains under the single heading of " perniosis ". According to Villaret (38), acrocyanosis results from venous hypertension due to a primary fault in the veins and venules. Claude and Baruk (6) report capillaro-venous atony but give no precise data. May, Bréant and Layani (31) examined the cardiac output in a number of acrocyanotics and found it to be normal, from which they concluded that there was no primary cardiac fault nor any obstruction to the circulation. Layani (19), in a separate paper, finds no arterial or venous spasm, but he thinks that acrocyanotics have a hypo-excitable sympathetic system, and that in the " cyanose de décliveté " there is a primary atony of the capillaries. In his book on this disease, however, Layani (18), having divided acrocyanosis into various types, considers " cyanose de décliveté " to be caused by the joint effects of gravity and vasomotor paralysis due to a supposed lesion of the cortico-striate nucleus. He is much concerned with endocrine and vasomotor considerations, and it is difficult to follow his arguments. A recent speculation is due to Paterson (34). He finds the respiration of schizophrenics to be shallower and quicker than that of normal subjects. When catatonia increases, the respiratory rate is said to fall below normal. He suggests that " failure of oxidation " may occur with cyanosed extremities. This is absurd, for cyanosis from such a cause, which would appear when there were about 5 grm. of reduced hæmoglobin per 100 c.c. of blood (Langdon-Brown (17)), would be general in type. Local cyanosis, like local inflammation, is always due to local causes.

In normal subjects local cutaneous cyanosis can readily be obtained by venous obstruction, as when a sphygmomanometer armlet, applied round a limb, is inflated to a pressure between systolic and diastolic. But in the case of normal cool skin, cyanosis is due to arterial or arteriolar constriction, which slows the blood-stream through the skin, resulting in dilatation of the capillaries, including the subpapillary venous plexus, owing probably to the effects of cold and an insufficient blood-supply. Such an area of skin readily becomes paler if raised above the level of the base of the heart, showing that there is no venous obstruction. T. Lewis and Landis (28) showed that this phenomenon could be demonstrated in a case of acrocyanosis. They also showed that

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light pressure on the acrocyanotic skin caused a white spot, which disappeared in a few seconds leaving no trace, showing that the blood had merely been displaced temporarily to adjacent parts of the skin. Thirdly, since local trauma produced a red area within the surrounding cyanosis, they concluded that there was an arteriolar obstruction. At the suggestion of the former of these authors all the cases reported here have been subjected to these three tests, and in each case these results have been confirmed.

The slowly disappearing white area caused by local light pressure was first noticed by Crocq (7), and its method of closing inwards from the periphery like an iris diaphragm is well known (18). The time for this closing to complete itself in the cases investigated here varied with the size of the spot, the level of the part in relation to the base of the heart, the degree of œdema and various other factors, and took from a fraction of a second to about a minute. The returning colour does not always appear to come exclusively from the periphery; a minute amount may come from the deeper parts, for the circulation to the skin has not entirely ceased in acrocyanosis, as is said to be the case in Raynaud's phenomenon (27), and a superficial prick or cut will cause bleeding.

The red area which follows local trauma in acrocyanosis is presumably due, as in normal skin, to a release of the H-substance which relaxes the arterioles. Lewis and Landis (28) noticed that the tint of the redness was very deep; probably this is because the cutaneous capillaries and venules are already widely dilated and so readily fill with fresh blood. It has been observed in the cases investigated here that the area of redness produced always greatly exceeds the area traumatized, and also that if the circulation to an acrocyanotic finger be occluded for 7 minutes by a rubber band, and then released, the reactionary hyperæmia, instead of ending at the mark made by the band, as in the normal finger, extends from one to two centimetres proximal to it. These two phenomena are presumably due to the ready diffusion of the released H-substance through the dilated minute superficial vessels to reach other arterioles. Allied to the above are the so-called cinnabar red spots, believed by Klingmüller and Dittrich (13) and Dittrich (9) to result from local arterial paralysis from summation of cold effects; they are simply areas of local arteriolar relaxation, probably sometimes due to local reactive hyperæmia as suggested by Haxthausen (10) or to any other cause of local inflammation, sometimes to trauma, but in all cases dependent on local release of the H-substance.

All these matters are readily explained by the hypothesis of T. Lewis and Landis (28) that in acrocyanosis there is an arteriolar obstruction. Release of the latter will be shown by the skin becoming bright red, and later normally pale as the capillaries and venules of the subpapillary venous plexus recover their tone. In the most severe cases, which occurred in mental defectives, relaxation is said never to occur, the feet remaining deep blue throughout the year, but even here local redness in response to trauma was obtained, though

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after a delay of a few minutes. In the ordinary severe case pallor is present only during a period of hot weather or rest in bed. The effect of shorter and local warming is to cause incomplete release, as shown by the following observations:

Observation 1.

P. P— (Appendix, Case 1), December 8, 1933, p.m. Room temperature 7° C. The subject stood and immersed his hands and wrists in a bowl of water at the level of his lower abdomen.

The temperature of the water was kept at 39–41° C.

The hands were deep purple and cold, there were a few small ulcers, probably chilblains, with no surrounding erythema. The forearms were cold, marbled and blue.

Hr.	min.	sec.	
		0	Both hands immersed.
	5	0	Colour of both a little deeper.
	9	0	Distal phalanges of left hand bright red.
	13	30	Right hand has paled a little.
	40	0	Both hands deep blue, except around the ulcers, where there is now a slight erythema. Colour of forearms unchanged. Hands withdrawn and dried gently; subject returned to ward.
3	25	0	Both hands deep blue, though a little paler than usual.

Observation 2.

Same case, January 1, 1934, p.m. Room temperature 7° C.

The hands were cold, and deep blue except for the digits, which were deep red. The forearms were cold, marbled and blue. The procedure was as above except that the water was kept at 44–46° C.

Hr.	min.	sec.	
		0	Both hands immersed.
	3	30	Metacarpal regions red, knuckles still blue.
	5	30	When the blood is gently pressed out of the blue patches it returns at once, but is redder, and then recovers its original blue colour.
	13	0	No blueness left except on ulnar borders of hands.
	14	30	The skin superficial to the principal veins on both forearms is warmer.
	15	30	The skin is pink for 7 cm. proximal to the water line.
	16	0	Both hands remain red.
	20	30	The redness has spread up the radial sides of the forearms along the veins nearly to the elbows.
	24	0	The colour of the hands is still deeper and a little bluer than normal.
	40	0	No further change; both hands withdrawn and dried gently.
	41	0	Colour unchanged. Subject returned to ward.
1	34	30	Dorsa of fingers, knuckles and ulnar borders of hands red. Dorsa of metacarpals a little fuller in colour than normal. Thumbs and palms normal. The hands are still warm.

January 2, 1934: Hands fully cyanotic.

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Observation 3.

G. S. M—, single, butter blender, admitted on November 27, 1932, aged 24; dementia præcox; shows waxy flexibility, automatic obedience, depression; stands about indifferent to his surroundings, rarely speaks and is impulsively violent. Marked acrocyanosis of his hands was noticed on January 20, 1933, and frequently since then. His blood was fully investigated, as in the appendix, and showed no abnormality.

December 12, 1933, p.m. Room temperature 9·8° C.

A less severe case than the above; he had kept his hands in his coat pockets all day; they were light mauve and slightly œdematous. The temperature of the water was kept at 38–42° C.

The procedure was as above.

Hr.	min.	sec.	
		0	Both hands immersed.
	2	30	Both hands reddening a little.
	4	30	Redness increasing.
	6	30	Left hand bright red.
	7	30	Fingers of right hand reddening more.
	10	30	Both hands paler. Forearms blue to water line, where they abruptly become bright red.
	13	0	Both hands bluer.
	18	0	Fingers of both hands going pink.
	23	0	Both hands redder; ulnar borders have remained bluish.
	35	30	Both hands bluish.
	41	30	Both hands mottled purple.
	42	0	Both hands reddening.
	43	0	Forearms still as at 10 min. 30 sec.
	43	30	Both hands going blue.
	46	0	Both hands reddening.
	46	30	Both hands withdrawn and gently dried; they at once went a uniform deep pink.
	49	0	Both still warm, going blue.
	51	15	Both bluish.
	52	30	Both going pink. Subject returned to ward.
2	30	0	Hands pale, but a little bluer than those of a normal subject.

In this observation the hands did not go permanently pink; their colour fluctuated simultaneously, recalling the phenomenon of "hunting" when the fingers are cooled in crushed ice (26).

T. Lewis and Landis (28) found by constructing colour-temperature charts and from the volume pulsation of the finger at various temperatures that the vessels could expand to near the normal, and so concluded that there was no structural change. These arguments are not conclusive. Recurrent spasmodic disorders of tubular structures are accompanied by muscular hypertrophy; e.g., congenital hypertrophic pyloric stenosis, and the hypertrophy of the intestinal, ureteric, or bladder-wall above a chronic partial or intermittent obstruction from any cause.

The cases of acrocyanosis observed here have stood or sat about, indifferent

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to the temperature of the parts, for long periods in almost any weather. They have been under constant supervision, and so have not been allowed to become extremely cold. Their extremities have in this way often been cooled to a moderate extent, at each fall in temperature the cutaneous arterioles have

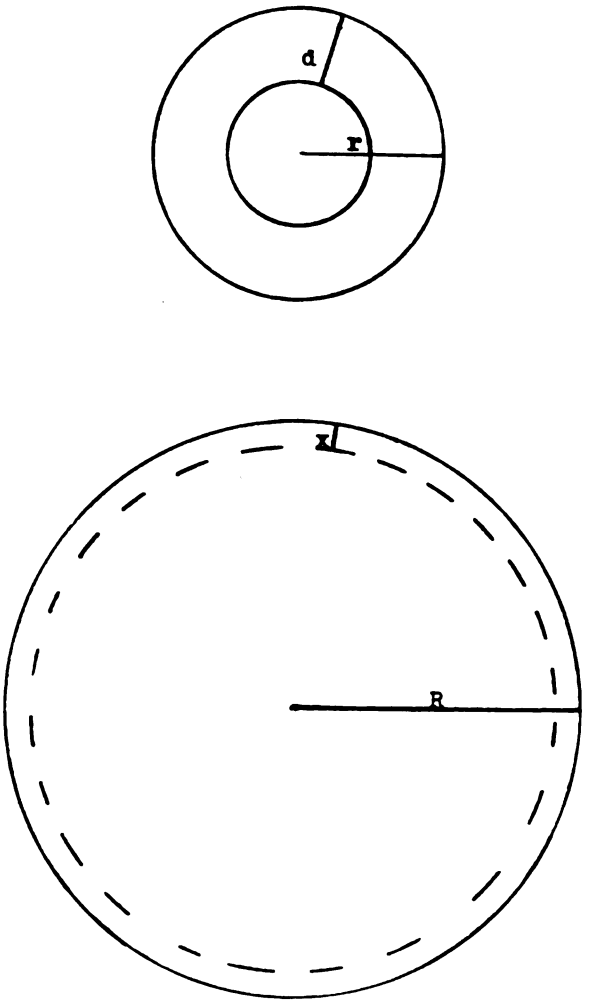

FIG. 1.

made the appropriate physiological response of contraction. Consequently hypertrophy of the muscle of the media of these vessels is to be expected. This involves some thickening of the vessel wall. Being elastic in nature, however, it will be negligible when the vessel is relaxed, as can be seen from the diagram (Fig. 1) and the following simple mathematical considerations.

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Let the radius of the lumen of a normal arteriole when contracted be r. then let the muscular part of the vessel hypertrophy so that its wall becomes elastically thickened by an amount d. The radius of the lumen is now reduced to $r - d$.

Then the area of cross-section of the thickening is—

$$\pi r^2 - \pi(r - d)^2.$$

Let the vessel now expand and its new radius be R, which is big compared with r.

If the thickening in the wall is now x,

$$\pi R^2 - (\pi R - x)^2 = \pi r^2 - \pi(r - d)^2,$$

since the area of cross-section of the thickening of the vessel wall must be constant, i.e.,

$$x^2 - 2Rx + 2rd - d^2 = 0.$$
$$\therefore x = R \pm \sqrt{R^2 - 2rd + d^2}.$$

But since r and d are small compared with R, their second order terms are negligible compared with R^2.

$$\therefore x = 2R \text{ or } 0.$$

But the solution $x = 2R$ is inadmissible and is obtained because—

$$(r - d)^2 = (d - r)^2.$$
$$\therefore x = 0.$$

i.e., when the arteriole is considerably expanded the thickening of the wall is negligible.

N.B.—In the above the density of the thickening in the vessel-wall is assumed constant, though in fact it will decrease on relaxation. But the error introduced will not be great enough to alter the result.

The above simple piece of reasoning is necessary for the proper understanding of affections involving spasm or thickening of the walls of the smaller blood-vessels. Ignorance of it has led to the "reactive hyperæmia test", long known (1), and recently described in exact terms by Pickering (35), being applied to differentiate obstructive from spasmodic affections of the peripheral vessels. His method is briefly as follows: The subject is kept warm in a room at or above 20° C., the limbs being heated by immersion for 10 minutes in water at 35° C., or by blankets and hot-water bottles. The limb is emptied of blood by elevation and its circulation abruptly stopped by inflating a sphygmomanometer cuff round the proximal segment of the limb to above systolic pressure. A bright red flush should reach the tips of the digits in less than 5 seconds, become maximal in less than 15, and fade quickly. The conditions laid down have been carefully followed. It was, however, found that warming of the limb by immersion in warm water was unsatisfactory, probably because

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the heat did not penetrate to the deeper tissues. Œdema of the part may also interfere with the validity of the test.

The following results were given by typical cases of acrocyanosis. The times are the number of seconds taken by the flush of reactive hyperæmia to reach the areas stated :

Case.	Pulp of fingers.		Pulp of toes.	
	Left.	Right.	Left.	Right.
1. App., p. 431 . .	2·0 .	2·2 .	3·8 .	2·2
R. R—, p. 423 . .	1·8 .	2·0 .	4·4 .	3·8
2. App., p. 431 . .	.. .	.. .	1·4 .	..
4. App., p. 433 . .	.. .	3·2 .	.. .	..
Case.	Dorsum of hand.		Dorsum of foot.	
R. R—, p. 423 . .	2·0 .	3·8 .	4·4 .	3·8
4. App., p. 433 . .	1·6 .	1·6 .	3·2 .	2·2
2. App., p. 431 . .	2·8 .	.. .	.. .	..
J. C. C— (acrocyanosis and arteriosclerosis)	.. .	1·2 .	2·4 .	..

They are all within the normal 5 seconds limit. Probably the test holds good in the case of structural diseases of the arteries, where such anatomical changes are rigid, but it is not valid in elastic thickenings of the smaller vessels. The skin from the dorsum of the hand and foot of these cases was sectioned, and in each instance showed much thickening of the media of the arterioles (pp. 422–427). Pickering (loc. cit.) states that in acrocyanosis the test is normal, but erroneously considers this to be due to the spasmodic, as opposed to structural, nature of the disease.

Pathology.

I. Histology.

(1) H. W. B—, a married labourer admitted on December 12, 1930, aged *22*; an epileptic imbecile. Physical examination showed no signs of disease ; the Wassermann reaction was negative. On November 7, 1931, he was found to have mild acrocyanosis of the hands and feet ; the systolic blood-pressure was 127.

He died in status epilepticus, and at the post-mortem on September 9, 1932, approximately 32 hours after death, a piece of skin was removed from the back of the right hand.

Sections show :

The veins of the subpapillary plexus are dilated. There is an increased amount of fibrous tissue in the cutis vera, where there is also slight hypertrophy of the medial coat in arterioles of about 40μ diameter. In the subcutis this hypertrophy is marked in arterioles of diameter 150μ and upwards. The largest arteriole seen just below the cutis has a greatly thickened medial coat and attains the abnormally large diameter of 400μ (Fig. 2).

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(2) R. R—, a single man, admitted on March 19, 1926, aged 24 ; dementia præcox ; continuously hallucinated, impulsive, shows waxy flexibility, stands about a great deal, indifferent to his surroundings. Physically he shows no sign of disease. Examination on January 19, 1932, revealed marked acrocyanosis of the hands and feet ; this was frequently observed before and since then ; blood-pressure 113/78. On January 9, 1934, biopsies were made, using

FIG. 2.—Cutaneous arteriole in acrocyanosis, showing thickening of wall. Case H. W. B—. × 400.

1% novocaine with adrenaline as a local anæsthesic, a few minutes being allowed for the fluid to absorb before removing the tissue.

Sections of skin of back of the left hand between the fourth and fifth metacarpals :

There is slight increase of fibrous tissue in the cutis vera ; the medial coat of arterioles of about 60μ diameter is considerably thickened.

Sections of skin of back of left foot 4 cm. proximal to the fourth interdigital cleft :

There is œdema of the cutis vera and subcutis ; just within the former there is thickening of arterioles of about 100μ diameter, the vessel wall being

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three times the average thickness, and the number of nuclei in the muscle being greatly increased, showing that proliferation has taken place (Fig. 3).

(3) S. M. O— (Appendix, Case 2).

(*a*) Skin from back of left hand, just proximal to knuckle of little finger, removed on July 26, 1935, using 0·75% cocaine hydrochloride with 1/100,000 adrenaline as a local anæsthetic.

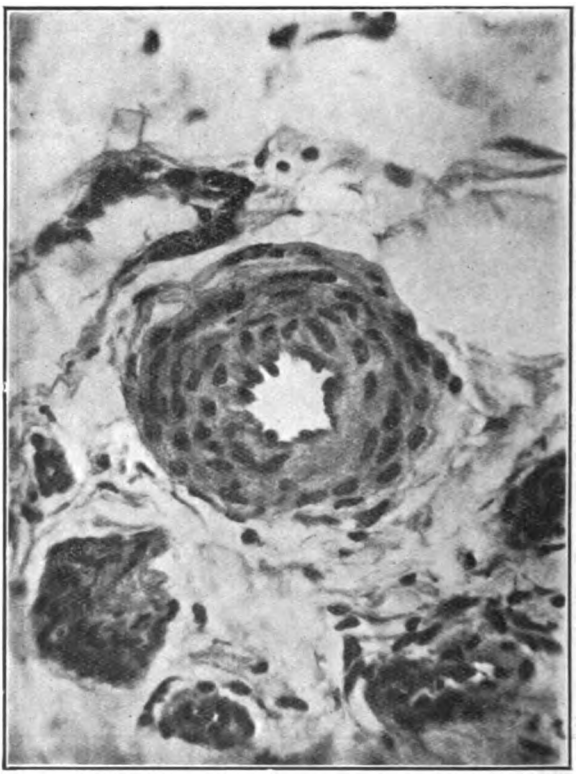

FIG. 3.—Cutaneous arteriole in acrocyanosis, showing thickening of wall. Case R. R—. × 400.

Section shows slight dilatation of the vessels of the subpapillary venous plexus, increase in the fibrous tissue and hypertrophy of the media of arterioles of about 30μ diameter with corresponding thickening of the venules in the cutis vera. The subcutaneous tissue is not shown.

(*b*) Skin from dorso-lateral border of left foot about 2 cm. proximal to fifth metatarso-phalangeal joint, removed as in (*a*).

One section shows increase of fibrous tissue in the cutis vera with hypertrophy of the medial coat of arterioles of 20 and 40μ diameter. The same change is seen in an arteriole of 30μ in the subcutis.

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Another section shows medial hypertrophy in an arteriole of 250μ diameter in the subcutis.

(*c*) Skin from dorso-lateral border of right foot about level of fourth interdigital cleft, removed on October 25, 1935, using 1% novocaine only as a local anæsthetic.

Section shows the subcutis, which is œdematous, and contains arterioles of 70 and 250μ diameter, the medial coat of which is thickened.

(*d*) Skin from dorso-ulnar border of right hand 1 cm. proximal to knuckle of little finger, removed as in (*c*).

Section shows dilatation of the veinlets of the subpapillary plexus, œdema and fibrosis of the cutis and hypertrophy of the media of arterioles of 300 to 500μ in the subcutis; these represent vessels which, lying just under the cutis, are very much bigger than those normally found.

(*e*) Skin from corresponding part of left hand, removed as in (*c*) except that adrenaline was added to the local anæsthetic.

One section shows fibrosis and œdema of the cutis and thickened media of arterioles of 30 to 40μ diameter.

Another section shows dilatation of the veinlets of the sub-papillary plexus, fibrosis and œdema of the cutis. In the subcutis there is œdema and medial hypertrophy in arterioles of 60, 200 and 700μ diameter, this last being enormously greater than any arteriole normally found in such a place.

(4) T. A. C— (Appendix, Case 4).

(*a*) Skin from back of left hand 2 cm. proximal to space between knuckles of middle and ring fingers, removed on July 30, 1935, using the same method as in (3*a*) above, and allowing ½ hour after the injection before making the incision.

Sections show dilatation of the subpapillary venous plexus, increase of fibrous tissue and some thickening of the medial coats of arterioles of about 50μ diameter in the cutis vera.

(*b*) Skin from dorso-ulnar border of right hand at level of fifth metacarpo-phalangeal joint, removed on October 11, 1935, using 1% novocaine only as a local anæsthetic.

Sections show that in the cutis vera there is fibrosis and œdema, and that the sub-papillary venous plexus is dilated; the medial coats of arterioles of 50 to 100μ diameter are thickened.

(*c*) Skin from lateral border of right foot 5 cm. proximal to the tip of the little toe, removed as in (*b*).

Sections show, in the cutis vera, considerable dilatation of the subpapillary venous plexus, œdema, fibrosis, and also thickening of the medial coats of arterioles, including some of 70, 90, 120 and 300μ diameter, this last being a vessel enormously greater than that normally found in such a situation. There is œdema of the subcutis with thickening of the media of arterioles of about 70μ diameter.

(*d*) Skin from part of left foot corresponding to (*c*) removed in the same way except that adrenaline was added to the local anæsthetic.

Sections show, in the cutis vera, dilatation of the subpapillary venous plexus, œdema, fibrosis, and also thickening of the medial coat of arterioles of about 100μ diameter. Other sections show œdema and fibrosis of the cutis vera and thickening of the medial coat of an arteriole of 50μ diameter; at the base of the cutis a similar change is to be seen in arterioles up to 150

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and 200μ diameter. In the subcutis is an arteriole of 300μ diameter, with thickening of the medial coat (Figs. 4 and 5).

Similar results were given by the examination of sections from 8 other cases of acrocyanosis; all were compared with the normal, and the degree of change was found to correspond with the severity of the disease. Sections

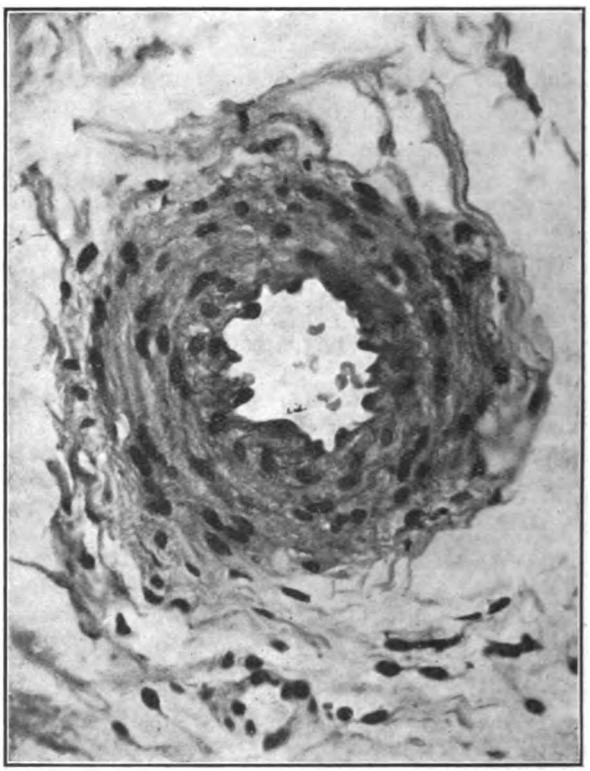

FIG. 4.—Cutaneous arteriole in acrocyanosis, showing thickening of wall. Case 4. × 400.

were stained with hæmatoxylin and eosin, and also with Weigert's elastin and van Gieson's stains in order to differentiate the medial coat. The essential change appears to be that the muscle tissue in the medial coat of all arterioles in the skin of from 30 to 700μ diameter is strikingly increased. This had been predicted by the considerations on p. 421 above, which also show that any such change must be great to be detectable unless the vessel is contracted.

It might be argued that in some of these cases normal arterioles merely in a state of spasm, perhaps from adrenaline, have been seen. The same changes, however, were seen in post-mortem specimens, e.g., (1), and in (3) and (4)

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corresponding portions of skin were examined (*c* and *d*), and adrenaline caused no appreciable difference. It was further found that in comparison with normal arterioles which might have been supposed to contract to such a size, with a wall three times the normal thickness, the number of nuclei in the medial coat was more than doubled, showing that a proliferation and not merely a hypertrophy had occurred. Again, in some cases the thickening was so great that the adjoining parts of the cutis and subcutis contained arterioles much larger than are normally to be found there.

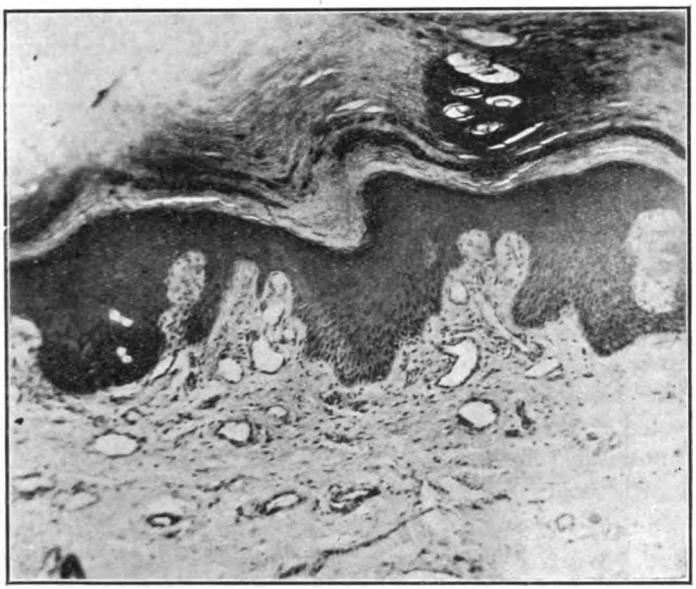

FIG. 5.—Dilatation of the subpapillary venous plexus and capillaries. Case 4. × 75.

The other histological changes—dilatation of the subpapillary venous plexus, fibrosis and œdema of the cutis vera and œdema of the subcutis are probably secondary to the local circulatory changes.

II. Hæmatology.

Layani (18) states that acrocyanotics show various blood disorders, especially prolongation of the coagulation time, up to an hour or more, change in the mode of coagulation caused by failure to retract properly on sedimentation, and lowering of the platelet count. These matters, and a number of others, have been investigated in some well-marked cases of acrocyanosis. In no case was any abnormality found; the results in 6 of these patients are given below in the Appendix.

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Prognosis and Treatment.

Acrocyanosis is a chronic disease; some cases do, however, improve as shown above on p. 414. These cases have kept themselves warm for a period of months. The mechanism of cure is easily understood from a consideration of the histological changes in the arterioles, for if the parts are kept warm the arterioles will remain in a relaxed state, and so the muscle of the medial coat will gradually atrophy back to its normal size. The prognosis is, therefore, poor in the absence of a favourable change of habit.

Treatment consists in keeping the patient as a whole, and especially the extremities, warm for months. In cases with œdema or ulceration, bed is necessary.

Related Disorders.

Erythrocyanosis crurum puellaris is now a well-known condition, and is considered to be due to the insufficient protection against cold given by modern female fashions of clothing. It is rare in men with their warmer nether garments. With the related *pernio follicularis* it was common in the female cases of acrocyanosis reported here. Klingmüller and Dittrich (13) have suggested that pernio follicularis is simply goose skin which has become permanent as a result of its frequent repetition. In this connection it was noticed that the dorsa of the hands and feet, though covered with hair, are never affected with goose skin or pernio follicularis. It is suggested that erythrocyanosis crurum puellaris is similarly a consequence of arteriolar narrowing, which causes capillary dilatation, owing to impaired nutrition of the walls of these vessels, because of their diminished blood-supply. The narrowing of the arterioles is a consequence of their frequent contractions in response to moderate cold, with resultant muscular hypertrophy as in acrocyanosis.

Erythrocyanosis crurum puellaris is often accompanied by local œdema. This œdema was noticed to be of a peculiar character; it may be present in the legs only, or legs and feet, leaving the ankles free; it is unusually firm, though it pits on continued pressure, and it shows little or no tendency to sink downwards. By means of its presence, accompanied by the "nutmeg grater" feeling due to pernio follicularis, erythrocyanosis can often be diagnosed between attacks, e.g., in the summer, when the skin is warm and of normal colour. It is presumably caused by transudation of fluid through the walls of the minute cutaneous vessels, which are rendered permeable as a result of their impaired nutrition, and is thus quite different from the œdema of congestive heart failure which is produced within the serous cavities and then gravitates downwards to the most dependent parts (Bolton (4) (5)). It is significant that of the 110 cases systematically examined above, 38% of the women, but only 7% of the men, had œdema of the lower limbs.

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Cutis marmorata is probably a mild form of erythrocyanosis seen in the trunk and upper limbs. Of two cases who showed it in the former area, one was an advanced case of syringomyelia with loss of temperature sense in this situation, and the other was in the habit of standing about in the open air regardless of the weather. The mottling disappeared if the patient lay down, showing that there was no obstruction on the venous side.

FIG. 6.—Newly-formed capillaries in the subpapillary region from a case of acrocyanosis. × 400.

Diffuse blueness of the face, especially the nose, lips, chin and cheeks, and sometimes the ears, was seen in 48% of 50 patients with acrocyanosis and only 10% of 60 without it. This blueness was of a similar shade and intensity to that of acrocyanosis. It is to be expected that exposed parts of skin should suffer from similar maladies, so that it is probable that the mechanism is the same.

The relationship of *chilblains* to acrocyanosis is doubtful, except that cold is an ætiological factor in both diseases. They were occasionally associated.

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This may merely be due to the chilling of the extremities which had been allowed to take place.

In *Raynaud's disease* there may possibly be a structural change in the affected arteries, perhaps a hypertrophy of the muscularis of the tunica media as in acrocyanosis. There is, however, one great difference between these two diseases: in acrocyanosis the circulation is only slowed during an attack, in Raynaud's disease it is stopped. This could be accounted for if in the latter the tunica intima were thickened. T. Lewis has envisaged this possibility, and states that "such anatomical evidence as we possess, though admittedly meagre, points to the presence of intimal thickening" (25), though this matter is still uncertain.

The *heart* and *great vessels* are *small* in many cases of dementia præcox, as noted by N. D. C. Lewis (20, 21)—a phenomenon frequently observed in this hospital. It is probably the physiological consequence of an exceptionally inactive life, and is thus like acrocyanosis a secondary condition. In this connection it is interesting to note that during an attack of acrocyanosis the radial pulse is small and there is a little difficulty in recording the blood-pressure, owing to the feebleness of the beats on auscultation or palpation, presumably because the rate of blood-flow through the extremities is lessened.

Discussion.

It has been pointed out above that local cooling is the essential ætiological factor in acrocyanosis, but it may be doubted if this alone would always cause the disease. General cooling of the body will cause failure of the extremities to react to excessive cold by the normal defensive mechanism of arterial and arteriolar dilatation (T. Lewis (24)). In these cases there was an indifference to cold, so that the whole body, as well as the extremities, was allowed to cool. Moreover, in dementia præcox, the condition typically associated with acrocyanosis, there is such apathy to bodily discomfort that the tips of the fingers are often charred from carelessness in smoking cigarettes. It is significant that, though cases of chronic encephalitis lethargica, under similar conditions to the others, have been observed here for some years, not one of them has developed acrocyanosis. Probably this is because, in spite of their motor disability, sensation has remained intact, and so they have not allowed themselves to become chilled. On the other hand, in the neuritic form of progressive muscular atrophy, in which sensation is impaired, coldness and cyanosis of the affected parts often occur.

Normal people whose extremities are exposed to cold commonly adopt some means of keeping the rest of the body warm, and thus preserve their local heating mechanism; e.g., officers on the bridge, sailors on deck and sentries do not stand still in cold weather, but march smartly to and fro; the Scots make up for their bare knees by wearing a warm kilt or plaid—in the

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absence of which the Boy Scouts' dress must be considered unphysiological for cold climates—and women commonly wear a warm wrap or fur to compensate for their scantily clad limbs.

The ætiology and morbid anatomy of acrocyanosis, demonstrated above, show that continued exercise of a muscle will cause it to hypertrophy, even in the media of the cutaneous arterioles of the exposed parts. Probably the same is true of other arterioles, and this may have some bearing on the development of arteriolar sclerosis and high blood-pressure. Again, attacks of spasmodic asthma are said to consist largely of spasm of the bronchial musculature, if this be so it follows that frequent repetition of attacks will lead to hypertrophy of this muscle tissue and to consequent chronicity of the disease. To cure this a prolonged period of complete freedom from attacks would seem to be necessary.

Appendix.

Cases whose blood was specially investigated. The men were patients at the Napsbury Mental Hospital and the women at the Shenley Mental Hospital.

Case 1.—P. P—, a single man of no occupation, admitted in November, 1930, aged 25; dementia præcox superadded to congenital mental defect. The history was that he had lived with his mother, who had been deserted by her husband. She developed ideas of persecution and later her son showed these same delusions. He lay in bed neglected at home. He can just read and perform simple calculations; speaks in a whisper. He is of puerile appearance, stands about always, totally indifferent to his surroundings, faulty in habits, does no work. Wassermann reaction negative. Since admission to this hospital he has been noticed to have marked acrocyanosis of both hands and feet. He has made no mental or physical change. Blood-pressure, 113/74.

January 18, 1935.

Hæmoglobin, 96%; red blood-cells, 4,930,000; colour index, 1·0; white blood-cells, 5,000.

Schilling hæmogram.—Basophils, 1%; eosinophils, 2%; myelocytes, *nil*; metamyelocytes, *nil*; banded neutrophils, 2%; multilobed neutrophils, 52%; monocytes, 2%; large lymphocytes, 8%; small lymphocytes, 33%.

Platelet count.—158,000 per c.mm.

Bleeding time (*Duke*).—2 minutes.

Coagulation time (*Dale and Laidlaw*).—2 minutes 26 seconds.

Calcium time.—Normal.

Prothrombin time (*Howells*).—Normal.

Fragility of the reds.—Hæmolysis commences at 0·6% and is complete at 0·5%.

Sedimentation rate.—Normal.

Case 2.—S. M. O—, a single man, formerly a clerk in the Admiralty, admitted February 19, 1934, aged 38; dementia præcox. There is a history of a previous attack in 1917, and he had been at home with his parents since 1921,

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having spent the last 6 years in bed. His appearance is like that of a mental defective; he is indifferent to his surroundings and unoccupied. Wassermann reaction negative. He always stands about, and has acrocyanosis of the hands and feet and a purple face. He develops chilblains on his hands in the winter. The feet are often œdematous. Blood-pressure, 112/90.

Fig. 7 shows his hands on March 12, 1935. Histological reports are given on p. 424.

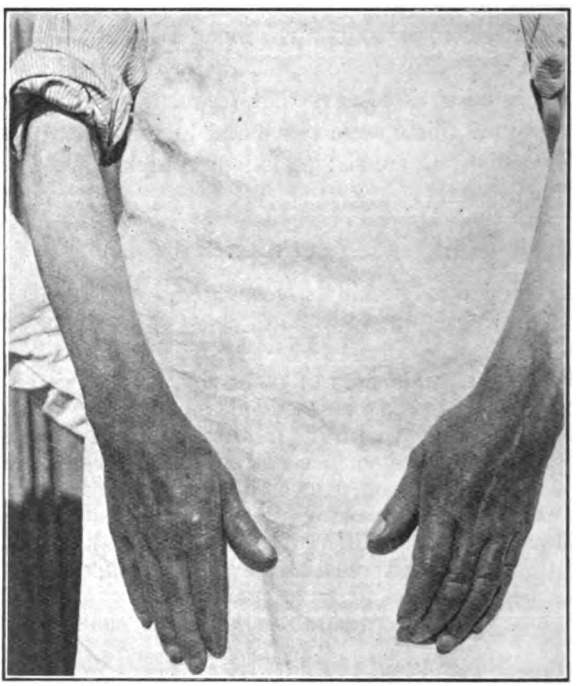

FIG. 7.—Acrocyanosis, the hands are dark blue, the colour fading gradually up the forearms. Appendix, Case 2.

January 18, 1935.

Hæmoglobin, 98%; red blood-cells, 4,860,000; colour index, 1·0; white blood-cells, 6,200.

Schilling hæmogram.—Basophils, 1%; eosinophils, 3%; myelocytes, *nil*; metamyelocytes, *nil*; banded neutrophils, 4%; multilobed neutrophils, 52%; monocytes, 7%; large lymphocytes, 9%; small lymphocytes, 24%.

Platelet count.—350,000 per c.mm.

Bleeding time (*Duke*).—4 minutes 30 seconds.

Coagulation time (*Dale and Laidlaw*).—2 minutes, 25 seconds.

Calcium time.—Normal.

Prothrombin time (*Howells*).—Normal.

Fragility of the reds.—Slight hæmolysis commences at 0·55% and is complete at 0·4%.

Sedimentation rate.—Normal.

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CASE 3.—C. W. C—, a married labourer, admitted on May 18, 1934, aged 32; dementia præcox; hallucinated, suspicious, grandiose. He rapidly demented and became depressed and unemployable, indifferent to his surroundings, immobile except for automatic shaking movements of the head and upper limbs. Wassermann reaction negative. In January, 1935, he was noticed to have fairly severe acrocyanosis of both hands and feet, with œdema of all these parts and chilblains on the hands. He had by this time become quite catatonic, and then gradually developed a generally flexed attitude. Blood-pressure, 140/108.

January 21, 1935.

Hæmoglobin, 95%; red blood-cells, 4,530,000; colour index, 1·05; white blood-cells, 5,200.

Schilling hæmogram.—Basophils, *nil*; eosinophils, 1%; myelocytes, *nil*; metamyelocytes, *nil*; banded neutrophils, 1%; multilobed neutrophils, 31%; monocytes, 5%; large lymphocytes, 34%; small lymphocytes, 28%.

Platelet count.—389,000 per c.mm.

Bleeding time (*Duke*).—5 minutes, 30 seconds.

Coagulation time (*Dale and Laidlaw*).—2 minutes 45 seconds.

Calcium time.—Normal.

Prothrombin time (*Howells*).—Normal.

Fragility of the reds.—Slight hæmolysis in 0·5%, complete hæmolysis in 0·4%.

Sedimentation rate.—Normal.

CASE 4.—T. A. C—, a single bank clerk, admitted on April 6, 1934, aged 25; dementia præcox, catatonic, has a delusion that he is controlled by telepathy, is faulty in habits and continually masturbates. Wassermann reaction negative. Since September, 1934, his face has been blue and he has had acrocyanosis of the hands and feet. He has had slight firm œdema of the lower limbs, and blistering, probably a cold injury, of the backs of the fingers. He keeps his hands stretched out in a curious attitude; both are often œdematous, the right more so. As is usual the blood-pressure is difficult to take when the hands are acrocyanotic, owing to the small quantity of blood passing through the brachial artery. In September, 1934, it was 90/72. On March 8, 1935, the left arm gave a reading of 103/88, and the right one of 119/88; both hands were blue, the left being more so, but there was œdema of the right hand only. Local pressure caused a white spot on the back of each hand; this disappeared from the left hand in 46 and from the right hand in 5 seconds. This patient has shown no further change, he rarely speaks, but is aware of all that goes on around him.

Histological reports are given on p. 425.

January 7, 1935.

Hæmoglobin, 94%; red blood-cells, 4,880,000; colour index, 1·0; white blood-cells, 9,600.

Schilling hæmogram.—Basophils, 1%; eosinophils, 2%; myelocytes, *nil*; metamyelocytes, *nil*; banded neutrophils, 3%; multilobed neutrophils, 66%; monocytes, 4%; large lymphocytes, 9%; small lymphocytes, 15%.

Platelet count.—225,000 per c.mm.

Bleeding time (*Duke*).—4 minutes.

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Coagulation time (*Dale and Laidlaw*).—2 minutes 45 seconds.
Calcium time.—Normal.
Howell's prothrombin time.—Normal.
Fragility of the reds.—Hæmolysis commences at 0·45% and is complete at 0·35%.
Sedimentation rate.—Normal.

CASE 5.—G. B—, a married woman, admitted on July 25, 1923, aged 31; dementia præcox, hallucinated, impulsive, suicidal, resistive to attention, usually walks backwards. She nearly always sits or stands about motionless and indifferent to her surroundings. Since 1925 she has needed occasional rest in bed for œdema of the feet; œdema of the hands was noted in 1926. In February, 1924, the extremities were noted to be cyanosed.

August 19, 1935: Face reddish; hands cool, appear normal, but the backs are yellowish, and after 12 minutes in water at 20° C. were in a typical acrocyanotic attack. Feet are cool and deep blue, showing severe acrocyanosis; the right big toe-nail has come off spontaneously; the left is atrophied and brittle; the feet and ankles are slightly swollen. There is slight pernio follicularis and firm œdema of the legs. Blood-pressure, 102/62.

January 29, 1935.

Hæmoglobin, 90%; red blood-cells, 6,060,000; colour index, 0·7; white blood-cells, 4,000.

Schilling hæmogram.—Basophils, 2%; eosinophils, 1%; myelocytes, *nil*; metamyelocytes, *nil*; banded neutrophils, 2·5%; polymorphonuclears, 66%; large lymphocytes, *nil*; small lymphocytes, 24·5%; large mononuclears, 4%. No abnormal reds or whites.

Platelet count.—340,000 per c.mm.

Coagulation time.—1st: 2 minutes 43 seconds. 2nd: 2 minutes 37 seconds.

Bleeding time.—4 minutes.

Volume index.—1·66 c.c.

Sedimentation rate.—

Mins.	c.c.	Mins.	c.c.
0	4·5	30	4·3
5	4·5	45	4·1
10	4·4	60	3·9
15	4·4		

CASE 6.—A. P—, a widow, cook, admitted on September 4, 1931, aged 58: demented, restless, resistive, rarely speaks, collects and eats rubbish, faulty in habits.

August 19, 1935: Face a little reddish, palms of hands reddish, backs brownish; after cooling in water at 20° C., as well as could be managed in the face of her resistance, they went into a borderline attack. She has not been wearing stockings, and the backs of the feet are exceedingly brown where not covered by the shoes; the legs are also sunburnt, and she has varicose veins. The feet were otherwise of normal colour when first seen; on cooling in air at 17·8° C. for 20 minutes they went into a mild attack of acrocyanosis. Blood-pressure, 126/79.

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February 1, 1935.

Hæmoglobin, 114% ; red blood-cells, 6,000,000 ; colour index, 0·9 ; white blood-cells, 3,400.

Schilling hæmogram.—Basophils, *nil* ; eosinophils, 2·2% ; myelocytes, *nil* ; metamyelocytes, *nil* ; banded neutrophils, 3% ; polymorphonuclears 56% ; large lymphocytes, 12% ; small lymphocytes, 24% ; large mononuclears, 3%. No abnormal reds or whites.

Platelet count.—620,000 per c.mm.

Coagulation time.—2 minutes 7 seconds.

Bleeding time.—4 minutes 30 seconds.

Volume index.—2·2 c.c.

Sedimentation rate.—

Mins.	c.c.	Mins.	c.c.
0	5·4	30	4·1
5	5·4	45	3·65
10	5·2	60	3·4
15	4·9		

Summary and Chief Conclusions.

1. Acrocyanosis is a true clinical entity ; it may affect the hands or feet or both. Its features are described.

2. It causes little disability except in extreme cases.

3. It may be latent, and a method of provoking attacks is described.

4. The mechanism of the reaction of normal skin to cooling by ice or iced water is shown to depend on an axon reflex.

5. The ætiology of acrocyanosis is frequent moderate cooling of the affected parts in conjunction with chilling of the body as a whole.

6. The age-limits are wide ; most of the cases in this investigation were between 20 and 45 years of age. There is no special sexual incidence. The disease is common in mental defectives and in the insane, being especially so in dementia præcox. There are thousands of cases in institutions in this country.

7. Its mechanism is a partial obstruction to the arterial blood-supply of the skin of the affected parts ; the implications of this are discussed mathematically.

8. There is no evidence of venous obstruction, and it is shown that in any similar case, anatomical changes in the arterioles can only be excluded by direct observation.

9. The obstruction is shown to be due to an increase in the muscle tissue of the middle coat of the arterioles of the cutis vera and subcutaneous tissue of the affected parts. It is not a mere matter of arteriolar spasm. Fibrosis and œdema also occur locally but are probably only secondary. There is no evidence of any pathological changes in the blood, nervous system or endocrine glands.

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10. The changes in the arterioles are the physiological consequences of the causes mentioned in conclusion 5.

11. In severe cases recovery from attacks may only occur after days of warmth. Treatment to be of permanent value involves practically continuous warmth for months.

12. The relationship of acrocyanosis to various other circulatory conditions and the bearing of this work on other diseases is discussed.

I wish to thank Sir Thomas Lewis for suggesting the subject of this paper, Dr. A. O'Neill, Superintendent of Napsbury Mental Hospital, Dr. W. G. Shore, Superintendent of Shenley Mental Hospital, Dr. H. E. Beasley, Superintendent of the Middlesex Colony for Mental Defectives, and Dr. E. Mildred Creak, formerly Director of the North-West London Child Guidance Clinic, for their interest and permission to publish cases, and the staffs of these institutions for their ready co-operation, especially Dr. W. H. McMenemey for his trenchant criticism and for the hæmatological investigations. I also thank Miss B. E. Stern for help with the mathematics, and Dr. W. G. Barnard, Chief Histologist to the L.C.C., for checking the histological findings.

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A NOTE ON SOME VASOMOTOR DISTURBANCES IN SCHIZOPHRENIA.

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Among the metabolic disturbances in schizophrenia there occur in many cases symptoms which are the result of vasomotor instability or upset. The causation of this vasomotor instability is not known, and it may in turn be due to undiscovered metabolic disturbances.

Cyanosed and œdematous extremities are almost constantly seen in cases of schizophrenic stupor which have continued for any length of time, but are much less commonly seen in cases of chronic schizophrenia which are not stuporose. In two of the cases in this series the patients were deteriorated, hallucinated and disjointed in talk, but remained in this state for two years before becoming stuporose. For the two years prior to passing into the stupor there were no signs of cyanosis, but two to three months afterwards their extremities were blue and œdematous. This cannot be accounted for by lack of exercise and underactivity, as massage and passive movements had no effect on the cyanosis in any of the cases.

Bullæ often appear on the cyanosed parts, which become mottled in colour and in some cases almost black.

One girl, aged 17, who was admitted in a stupor had marked cyanosis of the extremities, which resulted in gangrene of all her toes, definite lines of demarcation appearing. The toes were amputated, and the feet still remained cyanosed at the time of her discharge one year later.

Many of these cases, in addition, are subject to sudden attacks of collapse which, for want of a better term, may be called vaso-vagal in type. The patient becomes pallid, often vomits, the blood-pressure and pulse-rate fall (often to 50) and the temperature is subnormal. Within 10 to 15 minutes the attack passes off, the chief factor in aiding recovery appearing to be warmth. In all these patients there was no evidence of systemic disease in the heart, kidneys, etc.

The cyanosis of the extremities shows remarkable variations with the temperature, a cold spell of weather bringing on the cyanosis at once, together with bullæ and mottling of the extremities. In appearance, in the early stages, the affection resembles chilblains, and for this reason it was decided to investigate the calcium metabolism of 10 patients. The ages of the patients

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varied from 19 to 27 years, they were all stuporose and inaccessible and had been ill for from 4 to 9 years.

The estimations of serum calcium ranged from 8 to 14 mgrm.%, the average being 9·5. These estimations were within normal limits, even though they were on the low side in some cases, but there was no correlation between the calcium level and the degree of cyanosis present.

The patients were then given a series of 5 intra-muscular injections of 20 units of parathormone. This produced a slight rise in the serum calcium, the average rise being 2 mgrm., while the maximum was 3 mgrm. At the same time no change in the extremities was observed.

Following this, 6 intramuscular injections of 3 c.c. glucocalcium were given on alternate days. This is equivalent to about 4 c.c. of a 5% solution of calcium chloride. Again, only a small rise in the serum calcium, averaging about 1 mgrm., was noted, while again no change in the cyanosis was seen.

The fragility of the red cells and the viscosity of the blood were found to be normal.

The blood-pressure varied considerably from patient to patient, but was constant in each individual. The patients who showed the most marked degrees of cyanosis had blood-pressures of 95/55 mm. Hg and 140/95 mm. Hg respectively, while in the remaining patients it varied from 140/100 to 100/70 mm. Hg. There appears to be no constancy between the blood-pressure and peripheral cyanosis.

Krinsky and Gottlieb (1) investigated the venous pressures of schizophrenic patients and found they were similar to those of normal subjects, and they found no significant relationship between the peripheral cyanosis and the venous pressure level. Hoskins and Jellinek (2) found a more constant relationship of systolic and diastolic blood-pressures in schizophrenics than in normal subjects, and this suggested that their vasomotor responses to changing conditions are relatively sluggish.

The relation of the autonomic nervous system to the peripheral cyanosis is shown by the following case :

A girl, aged 21, in a state of schizophrenic stupor was admitted in 1929. The blood-pressure was 120/70, and she showed no physical disability, apart from cyanosed hands and feet. Four years after admission her feet became œdematous, showed red patches, and the toes were markedly discoloured, with a black spot on the third right toe. On April 30, 1933, because of impending gangrene, sections of the lumbar sympathetic nerves were removed on both sides. The feet, a few days after the operation, were normal, except that they showed postural cyanosis. Now, four years after the operation, the feet are quite normal, while the hands remain cyanosed and swollen.

Finkelman and Stephens (3) investigated the heat regulation in 50 schizophrenic patients, and found that they reacted to cold with a lower heat production than normal subjects, and that there was no continuation of an

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increased metabolic response after exposure, as occurred in the normal group. This suggests itself as a possible factor in causing peripheral cyanosis, the obvious defect being in the autonomic nervous system.

From all the above-mentioned points, it would appear that the fundamental disturbance on the physical side in schizophrenia is a clumsiness of adaptive responses to changed conditions, and that these vasomotor disturbances are due to dysfunction of the organism, together with an inadequate autonomic nervous system.

I wish to thank Dr. L. H. Wootton, Medical Superintendent of St. Ebba's Hospital, for permission to make use of the clinical material.

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SCHEMA FOR THE EXAMINATION OF ORGANIC CASES.

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Introduction.

IN a previous paper on general and focal symptoms localization in the brain was discussed as to its principles and limitations. In the subsequent investigations a method of examination has been worked out and is here presented. It is meant to serve a threefold purpose, which should be kept in mind when the scheme is applied.

(1) *Localization of Lesions.*

There have been great technical advances in brain surgery, but diagnosis has scarcely kept pace with them, at any rate so far as localization is concerned. It is true that radiographic methods have become more informative, but there are still many cerebral tumours which cannot be localized by these means, and in such cases the radiographic findings have to be interpreted, just as in other branches of medicine, in the light of clinical findings. The practical importance of localizing tumours and other cerebral lesions (such as wounds, hæmorrhages and scars) is incontestable. The new electro-encephalographic method introduced by Berger and elaborated by Adrian and others is not only a promising aid for local diagnosis, but confirms—a point which has hardly been emphasized—the view that there are circumscribed alterations of function in the brain. Although we hold no brief for the minute localization of mental functions, we have drawn attention in our previous paper to the existence of a number of definite correlations between psychological symptoms and localized lesions.

In the annotations of the following scheme we have given some cursory remarks on special localization. These are not intended to be exhaustive. Debatable points have been deliberately omitted.

However the symptoms in question are not found unless their possible existence is considered. This outline will, it is hoped, assist the examiner in finding them. Some scheme seems to be necessary, since so many of the symptoms are not likely to be observed unless especially sought for.

(2) *Refinement of Symptomatology.*

The application of such a scheme to the many cases with obvious cerebral damage which are in the hands of psychiatrists and neurologists should enlarge our knowledge of the symptoms of these conditions; for example when cases of general paralysis, arteriosclerosis and senile deterioration, or even mental deficiency, are examined with these methods, special disturbances can be isolated in a proportion of the cases. Dementia, as has been pointed out by

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Heilbronner, Liepmann and Pick, is not a simple and well-defined entity and is certainly not the negative of intelligence. These authors aimed at dividing dementia into psychologically well-defined disturbances. Successful though they were, there is still much work to be done in this field. Hence the proposal for a systematic survey of the available methods, such as is here tentatively offered.

(3) *Theory of Cerebral Function.*

Although it is not permissible to infer from symptoms what are the normal functions and where they are localized, a theory of cerebral function cannot be built up without taking into account the impaired functions seen in disease; for the elements of a function may become visible when the brain is damaged. That such a method of attack on the problem may yield valuable results has been demonstrated, e.g., in the investigations of Pick and Isserlin on agrammatism, of Goldstein and Poppelreuter on visual agnosia, etc. Here, too, the following outline may be of some value.

There are a number of *provisos* which it seemed desirable to bear in mind when drawing up such a scheme. Every method necessarily involves an underlying theory, which should always be tested again in the light of the facts; therefore the range of the examination should be as broad as possible. On the other hand it is impossible to examine everything. If one were to examine as much as possible one would pile up material without system and order. Therefore in this scheme we have tried equally to avoid the narrowness imposed by theory and the excessive latitude which a desire for completeness would entail. With regard to aphasic disorders T. Weisenburg expresses similar ideas as follows: "On the one hand, tests are bound to differ as long as theoretical differences exist; on the other hand, theoretical differences must exist, and must even appear greater, as long as the tests lack uniformity. Whatever the way out of the dilemma, it will undoubtedly be found through the study of large numbers of patients by comparable methods." This author notices that the main difficulty lies in deciding whether methods are comparable: he tries to solve this problem by using a number of the standardized tests of the psychologists.

Three objections can be raised against taking over these tests from normal psychology:

(1) There are hardly any *tests of special performances* (writing, drawing, spelling, etc.) standardized for adults. The intelligence tests for adults are, apart from all discussion about their general suitability, much too difficult for this purpose. Weisenburg has confirmed this again, and therefore turned back to mental tests standardized for children. He re-standardized them for adults. The norms thus obtained had to be corrected with regard to education, occupation, age and race. Considering the variability of each of these factors, practically every patient would have to be referred to a special standard.

(2) As regards the *arithmetical expression of the results*, it has still to be proved that the variations in the score are in each case proportioned to the difficulty of the task. Although this may be so in children or normal adults, it seems unlikely that the same relation should be valid in patients where the functions are disintegrated by structural damage. Such a disintegration is never homogeneous, and the re-arrangement of remaining functions alters the

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significance of each task. Furthermore one has to consider that the arbitrary procedure which is necessary to score the results, i.e., counting of errors and seconds, inevitably fails to give a full psychological account of the disturbed performance.

(3) The majority of the tests available aim at measuring *intelligence*. But a mere measuring of a reduction of the intelligence level cannot assist us in analysing the factors which qualitatively distinguish the intelligence of the dement from normal intelligence. Other tests devised for measuring special abilities (reading scales, etc.) seem better adapted for testing special defects. These scales, however, are standardized according to the development of the normal child. Using them here, one assumes that disintegration after cerebral lesions is the reversal of development. This has not been proved yet for many symptoms discovered since Jackson's time.

Consequently the following outline does not contain any standardized tests. Furthermore it abstains from scoring the results, since this, like the mere counting of positive and negative answers, could not tell us anything about the way the patient arrived at these answers. The type of mistake made, the devices by which the patient helped himself in the task and the accompanying emotions are of greater psychological importance than the results themselves. The time he takes has to be noted as an important factor, but here again one has to allow for affective and other concomitants.

The nature of the special disturbance present may be discovered by psychological analysis of the process by which the answer has been arrived at. Even a right answer may in this way be demonstrated to be the result of an abnormal mechanism which is compensated for. Such an analysis will sometimes require more detailed investigations and more specialized tests than can be provided in this outline, which in the main has been kept elastic, so that it could readily be adapted to the individual case. Additional tests may need to be employed, so that errors can be fully analysed and new allowance made for individual differences in education, occupation, sex, intelligence, etc. Moreover in organic cases general disturbances have to be taken into account (such as dimming of consciousness, impairment of memory). These are frequently the background against which can be seen the special functional anomalies. Only after a sufficient number of cases has been analysed in this way will it be possible to apply statistical methods.

Schema of Examination.

The following tests have been devised to supplement a routine neurological and psychiatric examination. They are less suitable for finding out if there is an organic illness. Their application is intended to reveal the special incapacity caused by the brain damage which has already been diagnosed.

It is best not to offer the tests in a set order, but to find out by taking a few sample tests which sort the patient does best, and then gradually work up to the more difficult ones (otherwise the patient may get upset and it will be impossible to assess his abilities).

It must be remembered that some organic patients are easily tired and that others do not show any fatigue at all. Both kinds of behaviour have to be noticed.

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It is not sufficient to state only whether the patient succeeded or failed in the test, but his actual performance and his answers should be recorded, whenever possible, in his own words. The patient's subjective experiences and his attitude towards them are of as much value in organic cases as in so-called psychological conditions. Hallucinations and illusions in all sensory fields have to be noted.

(1) *Mood.*

Is the mood labile? Is there perseveration of emotional expression? Is the emotional reaction exaggerated, perverted, lacking; is there a preference for one kind of emotion? Is the habitual mood in keeping with the premorbid personality? Do not overlook changes of personality caused by organic diseases like encephalitis lethargica.

A conversation on topics of emotional importance for the patient will give more information here than special tests. Shades of euphoria (*witzelsucht*, jocularity, lack of seriousness, etc.) are diagnostically more important than depressions, which are more frequently of secondary character. Various euphoric states have been put forward as localizing signs of lesions of the frontal lobe, the orbital region, the di- and mesencephalon. Lack of appreciation of the person's own symptoms, combined with euphoria, is an important symptom of a damage of the orbital cortex. Moral deterioration may result from irreparable lesions of the same region; also foci in the periventricular grey matter produce changes of personality. Irritability and apathy may be signs of general organic deterioration.

(2) *Orientation.*

Record even slight impairment. Distinguish the patient's orientation from his capacity to orientate himself in new surroundings. What aid does he use for it? Is there a common factor among the ways in which his disorientation shows itself?

Episodic loss of visual orientation has been described as a sign of fits originating in the occipital lobe. When disturbances of subjective time experience are present, a special examination of the capacity for estimating and reproducing time intervals may be indicated.

(3) *Attention and Adaptability.*

Is the patient's attention easy to attract? Can he concentrate his attention on his task for some period? Can he be distracted easily, and by what, or is he imperturbable to any stimulus? (Bourdon's test.)

The ease with which the patient's attention may be obtained varies according to the sensory field in which the stimulus is applied. There may even be differences between the various parts of the visual field (see p. 446). Remember that attention and concentration are influenced by fluctuations of consciousness. Distractibility of attention may be a symptom of organic impairment. ("Unselected responsiveness to stimuli," "hypermetamorphosis.")

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How far is the patient able to change from one topic or test to another? How far does this ability depend on his being interested and how far on the actual situation, especially the way in which the test is presented to him?

Perseveration, an extremely common symptom of organic disease, is often an obstacle to testing those cases. It can be present in various degrees, and may extend to words, phrases, actions or to the total behaviour. It is more likely to occur where the patient meets a difficulty, and can serve as an indicator of his shortcomings. Furthermore, an extreme "selectiveness to stimuli" may interfere with the patient's adaptability. The patient fails in everything that does not fit into the situation. He appears therefore quite undistractible (can be tested by Heilbronner's pictures, where the perseverating patient sticks to his first interpretation throughout the whole scale of the pictures). Some performances can be carried out when approached one way, but are impossible when attacked in another way ("focusing disturbance", "*Einstellstorung*").

(4) *Memory.*

Disturbances of remembering are always the most easily recognizable signs of an organic illness.

Besides the tests usually applied, detailed regard should be paid, if there is amnesia, to the dates of its beginning and its end. Was it complete while it lasted? Are there fragments or islands of memory?

It is important to observe the memory without the patient being aware of the purpose of the examination. Recollection of facts is often better when it is incidental than when it is deliberate. How much does the amnesia cover the time before and after the lesion which caused it? Did the amnesic period coincide with a disturbance of consciousness (retrograde and anterograde amnesia)? Are there special memory defects—e.g., for dates, numbers, visual or verbalized impressions, etc.?

Non-language tests of memory are of special importance in all cases where there is the slightest suspicion of an aphasic disorder (vide below).

Does the patient fabricate, does he do so spontaneously or in response to suggestion only?

(5) *Use of Numbers.*

Can the patient read and write figures and numbers of two or more digits? Has he a general concept of numbers and measures? (inhabitants of London, are there thousands or millions? Height of a man, inches or feet? Weight of a man, 5 stone or 50? etc.).

Can the patient add, subtract, divide or multiply? Let him do it on paper as well as in his head, with plain numbers as well as in concrete problems. Can he count objects, recognize dots on dominoes and similar groupings? Can he guess without counting how many matches are lying in front of him? Can he divide them into two or three equal groups? Let him compare a few big objects with many small ones of the same kind (buttons). Can he count money?

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While disturbances in writing single figures generally belong to the field of agraphia, mistakes in the sequence of figures and loss of the position value are a characteristic arithmetical disturbance *sui generis*.

Multiplication tables being most automatized are of little use for testing arithmetical ability. Counting of objects one by one is entirely different from recognizing dots arranged in a group at one glance. This again is different from estimating the size of an unorganized multitude. Disturbance of all these performances may be observed with lesions of the occipito-parietal convexity.

(6) *Speech.*

How is the patient's *spontaneous speech*? Does he articulate correctly? Does he hesitate or stammer? (which can be a sign of aphasia). Notice his modulations and the inflection of voice. Is his voice monotonous or does it fade away? Can he repeat words or sentences? (words he understands and unknown words like Artaxerxes, Lysistrata). Can he recite automatic word series? (days of the week, months of the year, a prayer or poem).

Notice the grammar in his spontaneous speech; if necessary ask him to tell a story. Test his capacity to conjugate or parse. Ask him to build up a sentence using three given words: hunter, fox, field; child, scissors, hurt; gun, soldier, battle.

Does he produce any paraphasic utterances?

Does the patient *understand* single words? (e.g., objects: Give me the pencil! Where is the door?). Does he understand simple or more complicated sentences? (orders: Open the window. Take the chair and put it in the corner. Open your mouth, shut it again and point to your right eye). [Memory disturbance and apraxia may interfere with this.] Can he distinguish words which sound alike and can he use them correctly? (father, farther; breeze, breathe; boat, bought; dumb, thumb; task, tusk; collar, colour; coal, cool).

Does he notice and can he correct mispronounced words? Can he find the right words? Names of objects and pictures, abstract nouns, proper names? (Colour-names: see p. 447.)

In cases of severe disturbance: how far are emotional utterances preserved? (swearing, interjections). Can he indicate the number of syllables in words which he cannot utter? (Lichtheim's test of inner language). Is any former command of *foreign languages* impaired? Is his *vocabulary* poor? (Tell me the names of flowers, pieces of furniture, etc. Find opposites to given words such as good, outside, quick, empty, many, friend, etc. Find rhymes to words like fair, round, patch, etc.)

Since all the tests mentioned earlier require for their performance full command of speech, it is advantageous wherever there is any suspicion of aphasia to examine for it before doing other tests. Aphasia is often overlooked, or is mistakenly regarded as due to confusion, dementia or hysteria. By means of the above scheme only a coarse outline of the patient's speech impairment can be arrived at.

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For detailed analysis, systematic examination must be carried out as proposed by Heilbronner, Feuchtwanger, Weissenburg and others.

This scheme has been made as elastic as possible, since this is more necessary in the field of aphasia than anywhere else. The variability of the findings and the adaptability of the patient can yield more information than an artificial constancy in the result.

A series of tests such as employed by Head has not been recommended here; with them it would be difficult to allow for individual differences in linguistic gift and education, and to take into account the extent to which the patient's ability to learn is preserved. Moreover the tests do not cover the whole, wide field of the use of language.

It is generally agreed that the brain lesions which produce aphasia are located in the left hemisphere when the patient is right-handed and vice versa. Furthermore foci producing expressive (motor) aphasia are to be found in the anterior parts of the brain, those producing the receptive (sensory) forms further back. The finer localization of other subdivisions of aphasia is still very much under debate. Just as in assessing the results of verbal tests the patient's previous level of intelligence must be taken into account, so in using intellectual tests due regard must be paid to the speech disturbance, since this might notably interfere with the intellectual process—much more so, indeed, than any other focal lesion would.

When speech is disturbed the following *non-language tests* may be employed as substitutes for the methods suggested before: Pintner-Paterson substitution test; Knox's cube test; Porteous mazes; sorting of objects or of small pictures of objects; form-colour-test (sorting cardboard pieces of various shapes, sizes, and colours); portrait test (show the patient a portrait and then ask him to find it when it is mixed with eight others).

The interpretation of the results of these non-language tests is only possible after carrying out the tests for agnosia and apraxia.

(7) *Visual and Related Functions.*

Measure first the visual acuity and record the field of vision either by rough testing or on the perimeter with different colours. Scotomata? blinking reflex? optokinetic nystagmus? reaction to strong stimuli, e.g., bright light?

Are there any visual hallucinations? illusions? (note alterations of size, shape and colour). How are moving objects perceived? (too slow, too fast, stationary). If necessary, test with stroboscope or cinema. Is there a consistent distortion of visual space?

Visual attention.—Ask him to name or to find various letters or numbers of objects drawn all over the surface of a blackboard. Does he neglect those in any particular region?

Visual memory or imagery.—Get him to show the size of a well-known object with his hands (a cat, a horse, etc.) Does he remember the colour of objects mentioned to him? Get him to describe his house or the situation of his bed in the ward. Wertheimer's test (two isosceles right-angled triangles of the same size, cut out from paper, are shown to the patient, who has to

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indicate what figure would be formed by putting these two triangles together, side by side, in various ways. The answer can be a square, a lozenge, or a larger triangle).

Can he recognize, when he sees them, objects, gestures and simple happenings? Can he name them? Can he recognize pictures of single objects and scenes? Is he able on request to pick one out of a large number of pictures (or letters) spread on the table?

Can he recognize and name simple geometric patterns? (triangle, square, circle). Can he disentangle them in Abelson's test?

Can he connect two given dots by a straight line? Can he find the middle of a straight line or of a circle?

Ishiwara's test for colour-blindness.

Perception of *colours*? Can he name them? Can he sort out colours on request (Holmgren's wools)?

Can he tell the time from the *clock*? Can he set the hands of a clock to a given time?

Can he use a *map*? Can he find the points of the compass? Can he point to visible objects in different parts of the room? Can he demonstrate and name *directions*—above, below, horizontal, perpendicular, etc.?

Can he guess distances? Can he see perspective correctly?

Methods of examination which usually are regarded as neurological have been put at the top of this list, since their results are often modified by central visual disturbances, in which case the responses obtained are much less constant than they are after lesions of the more peripheral part of the visual apparatus. Disturbances of specific, i.e., here visual, attention (such as "*Seelenlähmung des Schauens*", Balint) and also specific fatigue can produce phenomena which may be mistaken for hysterical ones, because of their lack of constancy. One-sided absence of the blinking reflex or optokinetic nystagmus may be due to hemianopia or to partial disturbance of attention. Inadequate response to optic stimuli (even to sudden and strong ones) can be an initial or residual symptom of visual agnosia.

In spite of constitutional differences with regard to the faculty of visualization, a certain amount of visual imagery can be expected from everyone. The distinctness and range of images can be interfered with by cerebral lesions; in particular, the ease with which they are recalled can be diminished. Purely subjective anomalies of vision such as micropsia, macropsia and other distortions cannot be described by the patient except by comparison with such visual memory as he has preserved.

By testing with pictures minor disturbances of visual perception may be revealed in patients who are able to recognize objects. Others do well with pictures of single objects, but fail in understanding a scene if this requires them to bring the parts of the pictures into proper relation (comprehension-disturbance, Pick). Children's toy books can be used for examinations of this kind.

Some among the tests of this chapter which could have been given under other headings as well are mentioned here for the sake of convenience. For instance, impairment of colour-names really belongs to aphasia, but may occur independently. Inability in setting a clock may be a sign of apraxia, or secondary to disturbances in the use of numbers. Failures in spatial tests can be of various origin: recognition of the points of the compass depends on the distinction of left and right (see below). Pointing may be disturbed by apraxia. Immediate

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orientation in space is dependent on the " body-image ". This central representation of one's own body can be disturbed by a lesion of the parietal lobe as well as by damage to the vestibulo-cerebellar system.

Roughly speaking, disturbances of the visual perception and related functions such as those mentioned are to be found after lesions of the occipital lobe. Perceptual anomalies of a lower level occur after lesions of the calcarine area whereas more complex disturbances appear when wider areas of the occipital lobe, including its convexity, are involved.

Can he *read* letters, words, sentences—aloud and silently? Is there a difference between writing and print? Does he keep to the line? Does he understand written sentences, e.g., requests? Does he find out mistakes in spelling? Can he read words in which single letters are struck out?

All tests of visual perception may be increased in difficulty by shortening the time of exposure (tachistoscope).

Alexia may occur with, and be dependent on, aphasic disorders of all kinds. Aphasics who cannot read aloud can still often understand what they read silently o themselves. Of special interest is the interdependence of alexia and agraphia, though both can occur separately. Patients may be unable to recognize single letters, or they may be able to do so, but unable to read words as a whole.

Mild alectic disturbances are discovered only when the test is made harder, e.g., by giving words in which single letters have been struck out. This can be an insuperable obstacle for an alectic patient, although a normal person hardly notices the difference. Lesions in cases of alexia may be found either in the parietal or in the occipital lobe.

(8) *Auditory Perception.*

Does he recognize *noises*? (whistling, rattling, banging, barking, mewing, etc.). Does he recognize objects from sounds? (match-box, coins, brush). Reaction to strong auditory stimuli? (shot of a pistol). Can he point towards noises coming from different directions? ("acoustic localization ".)

Understanding of spoken *language*: see under (6).

Music.—Is he able to recognize tunes, to repeat them, to sing spontaneously? Use of musical instruments if possible. Can he repeat a *rhythm*, knocked out to him on the table? Can he roughly estimate intervals of time?

An exact examination of the peripheral hearing is of the same importance as the examination of visual acuity and visual field. The interaction of peripheral and central disturbances in the province of hearing are closer than in seeing, but much less known, since the methods of distinguishing them are still somewhat defective.

Central disturbances of auditory perception are to be observed after lesions of the temporal lobes, the gyri Heschl preferably. Double-sided lesions are probably necessary to produce permanent defects.

(9) *Tactile Perception and "Body Image".*

Does the patient recognize materials and objects by feeling? Does he recognize numbers written on his skin? Can he describe the direction of a line drawn on his skin? Can he imitate postures of his limbs from one side

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to the other and imitate the examiner's postures? Can he point to parts of his own body or other people's? (Head's finger-nose-ear-test).

Does he recognize and name and demonstrate single fingers? Can he distinguish between right and left? Can he do this on a person sitting opposite to him?

Is there any anomaly of the awareness of his own body or parts of it?

The routine examination of superficial and deep sensation must precede the application of these tests. In all doubtful cases standardized stimuli should be used, such as von Frey's set of hairs, or compasses. Not only quantitative anomalies, but also qualitative variations of sensation have to be recorded, such as are to be observed especially after thalamic lesions. The recognition of surfaces of material, e.g., silk, velvet, paper, wood, can be disturbed without impairment of the common skin sensation. The same is true with regard to the recognition of shapes and objects. Numbers written on the skin are generally recognized independently of their orientation in space. These functions are found disturbed after lesions of the post-central gyrus and the adjacent parts of the parietal lobe.

When using the finger-nose-ear-test, one has to bear in mind that the results depend to some extent on the patient's memory and speech. Sometimes the disturbances appear only when the patient has to cross the mid-line of his body with the hand to carry out the test. Finger agnosia may be found as a relatively isolated disturbance, often together with difficulties in distinguishing right and left. Both are due to lesions in the lower parietal lobe, probably of the left side. Disturbances of the body image have been observed as loss of perception of a part of the body (autotopagnosia), wrong localization of it, symmetrical displacement (alloæsthesia), loss of self-perception of pareses and other disturbances of function (anosognosia).

(10) *Motility.*

Does the patient's motor behaviour as a whole show anything exceptional with regard to amount, tempo, co-ordination and harmony of movements?

Observe the patient's behaviour, especially when he carries out movements requiring some skill, such as throwing and catching a ball, jumping over an obstacle, carrying a tray with several objects.

Is there any anomaly in spontaneous *facial expression*? Can he produce facial expressions on request—smile, look astonished, etc.? Can he nod "yes" and "no", whistle, click his tongue, pout, lick his upper lip, close one eye? Can he make *gestures* correctly? (beckoning, military salute, threatening, waving good-bye. To be tested with each hand separately).

Single movements of joints and limbs might be disturbed without paralysis: moving of single fingers, of the toes, opening and closing the hands, doing "physical jerks" with arms or legs. Notice especially synkineses of the other side!

Can he *imitate* all the movements and postures demonstrated to him? Can he perform activities on his own body correctly? (scratching, twisting moustache).

How does he perform *purposive activities* with objects? (using scissors, a

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key, threading a needle, putting a letter into an envelope and posting it, combing his hair while holding the glass, etc.).

Try activities with particular difficulties in space: putting on spectacles, forming a string into a bow, folding a napkin.

Purposive make-believe activities (knocking at a door, using a hammer and nail, using a typewriter, playing the violin).

Poverty as well as abundance of spontaneous movements can be a symptom of organic disease. The disturbances may be noticeable in the whole body or be limited to parts of it. They may be caused by lesions in various areas of the brain. Slowness and rapidity of movements, well known after lesions of the extra-pyramidal system, may be produced by lesions of a higher level as well. The same holds true for tic-like, choreiform and similar hyperkineses. Their diagnostic value can be assessed only within their particular setting. They can be of organic origin, even if they appear only under the influence of emotions.

There is a special type of apraxia, limited to the face. It is characterized by the inability to carry out expressive movements deliberately, while the automatic expressive movements are preserved.

Mild cases of apraxia may appear just "clumsy", severe ones confused. Apraxia may be confined to one limb or one side of the body. Purposive make-believe activities reveal the disturbance more easily than ordinary activities with objects do. Lesions in cases of apraxia are to be found in the corpus callosum, the parietal lobe and, possibly, in the frontal lobe.

Constructional activities.—Can the patient imitate patterns of matches, blocks or mosaics? Free construction without pattern? Does he recognize his mistakes?

Drawing.—Can he draw on request a cross, triangle, house, man or elephant? Can he copy simple drawings? Can he trace?

Writing.—Can the patient write down letters, words, sentences dictated to him? Can he write his signature? In cursive and block letters? Can he answer a question in writing? Can he copy from print? Guide his finger so that he writes letters and ask him to read them. Can he put together single wooden letters to form a word?

Constructional inability is relatively independent of other apractic disturbances. As a rule, disturbances in drawing and in constructing occur together. The inability is generally so striking in cases of this kind that the usual standardized performance tests are of no use; they are much too difficult. Only such gross inability is of diagnostic value, since the average person's performances in these tests, especially in drawing, is poor. The tests are especially useful, since they are independent of vocabulary and memory. The lesions in cases of constructional disturbances are to be found in the lower parietal lobe, near to the occipital lobe.

Disturbances of writing are most commonly combined with disturbances of language and secondary to them. All types of aphasic disorders are reflected in the patient's writing; but there are also isolated agraphias. Agraphia may interfere with the writing of the single letter, construction of words or the arrangement of the lines. The signature being a fixed stamped pattern is often the *ultimum moriens*. Figure-writing, too, plays a particular part (see above). Some patients, though unable to write, preserve the ability to draw letters. Disturbances of writing can be observed independently of reading disturbances.

Lesions in cases of agraphia are distributed over the brain exactly like those in aphasia. Besides, there are parietal lesions in cases of pure agraphia.

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Conclusion.

In conclusion the authors wish to mention that they are well aware of the preliminary nature of such a schema, and they hope that its application and discussion may lead to its improvement. Incomplete as our present knowledge in cerebral symptomatology is, it may be expected that the scheme will grow richer and more precise as this increases. Moreover, it has to be remembered that in each individual case the examiner may be confronted with individual problems exceeding the schema. The insufficiency of the schema in such cases may disclose the point where further work is called for.

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THE IRON, COPPER AND MANGANESE CONTENT OF THE HUMAN BRAIN.

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THE observations recorded in this paper form part of an investigation into the mineral constituents of the central nervous system both in normal and pathological conditions.

Iron.

Iron is a respiration catalyst, and recent work has tended to emphasize the importance of this element in the brain (1). The brain cortex is very rich in cytochrome, the iron-containing pigment which plays an intimate part in cellular metabolism (2).

Exhaustive histochemical observations on iron distribution in pathological states have been made by Spatz (3), and confirmed by others (4, 5). The quantitative distribution, however, has not yet been completely studied, and there is a wide divergence in the results recorded (6–11).

The first part of this investigation consisted in the estimation of the total iron in the various parts of the brains of a large number of " accident " and pathological cases.

> The brain was washed as free from blood as possible, and portions of the cortex, white matter, cerebellar cortex and corpus striatum (putamen, nucleus lenticularis, and nucleus caudatus) were severally isolated. The dried tissue was ashed, and the iron estimated in a solution of the ash by the colorimetric method of Lyons (12). Recovery 90%. Error for duplicates ± 6·8%.

On this dry basis, the content of the cortex was the same as that of the cerebellar cortex; the white matter contained about half as much iron as the cortex; and the corpus striatum contained a large excess over that of any other part.

Of the various pathological cases, marked differences from the average occurred only in the G.P.I. cortex, many of which gave higher values. The results obtained in this way are given in Table 1.

The cases of general paralysis whose brains were submitted to the Central Laboratory for investigation are those of patients (*a*) who have died without treatment, (*b*) who have died during a course of malaria or tryparsamide treatment, (*c*) who showed no signs of remission after treatment, (*d*) whose reaction to treatment was such as to cause a complete arrest of the progress of the

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TABLE I.—*Distribution of Total Iron in the Human Brain (mgrm./100 grm. dried tissue).*

		Fe.	n.	σ.
" Accident " and pathological cases :	Cortex . .	22·6	35	3·16
	White matter	12·6	30	2·12
	Cerebellar cortex	22·2	31	4·25
	Corpus striatum	55·5	35	13·2
G.P.I. :	Cortex	29·0	19	4·62

n = number of cases estimated. σ = standard deviation.

disease, but who had to be retained in hospital on account of an irrecoverable dementia. These latter cases had died of other non-paralytic disorders at various dates up to ten years from the time of the treatment. In the majority of such cases of total arrest of the disease, histochemical examination reveals no signs of the lesions of general paralysis other than nerve-cell degeneration. All cases diagnosed as G.P.I. gave the typical serological and humoral reactions on examination before treatment.

It was found that the total cortical iron in those cases where histochemical examination revealed characteristic signs of G.P.I. in the brain was significantly higher than in those cases where there were no signs of active disease :

	Fe.	n.	σ.
No signs of active G.P.I. .	26·0	6	5·11
Signs of active G.P.I. . .	32·3	13	4·37

The results were compared by the " t " method of Fisher (13) for comparison of two means. In this case t = 2·60, which is significant.

The cases were then classified according to whether or not the histochemical examination revealed a characteristic deposit, usually assumed to be iron, in the cortex. The water content was determined (brain not washed) ; and the iron estimated in mgrm./100 grm. fresh tissue. It was found that those cases having a histochemical deposit were significantly higher (t = 3·80) than those having no deposit :

	Fe.	n.	σ.
No deposit . .	3·74	10	0·56
Deposit . . .	5·05	17	0·98

With three exceptions, in 37 G.P.I. cortexes examined histochemically and compared with the general appearance of the brain, there was no histochemical deposit when there were no characteristic histological appearances of general paralysis ; and there was a deposit when there were characteristic signs of G.P.I. The exceptions were : two cases showing no deposit where there was a slight appearance of G.P.I., and one case having no appearance of G.P.I.

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where a deposit was observed in the cells (or ? neuroglia) and not in the capillaries.

The next step was to ascertain whether this excess of total iron found in the G.P.I. cortex was due to an excess of blood (50 mgrm. Fe/100 ml. blood), or whether the excess was in the non-hæmatin or "available" portion.

There are several methods for estimating "inorganic" (11), "available" (14, 15), or "ionizable" (16) iron in tissues, but there is no evidence that, by any of these methods, all the non-hæmatin iron is extracted; and, in the case of the Tompsett (11) technique, this cannot be so, because I have obtained very much higher values by leaving the tissue overnight in contact with the reagents, than for 30 minutes, as advised by Tompsett, maximum values being attained in about 24 hours. In this time it is probable (as a result of experiments not detailed here) that, in the case of the cortex and corpus striatum, all the non-hæmatin iron is extracted; and in addition a very small amount of blood iron, insufficient to affect the results significantly, i.e., significant variations will not be due to blood. I have given the results obtained by the Tompsett technique, leaving the tissue in contact with the reagents for 30 minutes and 24 hours, and have referred to these respectively as representing "inorganic" and "available" iron.

Table II shows that the average "normal" distribution of total and "available" iron per 100 grm. fresh tissue is approximately equal throughout the cortex, white matter, and cerebellar cortex; with a large excess, mostly "available", in the corpus striatum, and particularly in the pallidus and substantia nigra.

TABLE II.—*Distribution of Total and "Available" Iron in the Human Brain (mgrm./100 grm. fresh tissue).*

	$H_2O\%$.	n.	σ.	Total Fe.	n.	σ.	"Available" Fe.	n.	σ.
Cortex	83·0	18	1·78	3·62	19	0·62	2·81	7	0·63
White matter	68·5	16	2·50	3·77	17	0·48	2·59	6	0·76
Cerebellar cortex	81·5	13	1·88	3·68	14	0·49	2·83	5	0·79
Corpus striatum	79·4	23	1·72	11·50	28	2·35	9·92	8	2·14
Pallidus	73·6	4	2·15	20·60	5	3·51	22·10	4	9·00
Substantia nigra	70·8	3	2·64	20·86	3	6·50	26·80	1	
Thalamus	75·5	7	2·68	6·26	8	2·29	5·96	3	0·68

Estimation of the "available" iron in the cortex of the G.P.I. gave the following results:

	Fe.	n.	σ.
No deposit	2·69	5	0·368
Deposit	4·46	8	0·506

There is a significant ($t = 5·96$) excess of "available" iron in those cases where a deposit was observed histochemically.

In Table III details are given of those cases where the total and "available" iron was determined on the same sample. In the case of the G.P.Is. there was also a significant ($t = 5·97$) excess of "available" iron in those cases

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where a deposit was observed histochemically. These results show that deposits of iron demonstrated by histochemical methods are not the expression of a redistribution of the normal cellular iron content, but represent iron deposition from extracellular sources. The frontal, medial and occipital regions of the cortex were, in several cases, taken for analysis. In the case of the G.P.Is. the frontal region sometimes contained most iron; and, where the three regions were analysed, the value for the frontal region only is cited in the table. The value for the pallidus in Case 186 has been given, because this part is specifically affected in CO poisoning (5); the iron content in this case was normal.

TABLE III.—*Total and "Available" Iron in the Cortex of the G.P.I.* (*mgrm./100 grm. fresh tissue*).

	Case No.	Total Fe.	"Available" Fe.	
"Normal"	186	3·28	2·62	CO poisoning (pallidus 20·02).
	209	3·57	2·65	Diabetes.
	228	3·76	3·24	Accident.
G.P.I. (no deposit)	196	4·12	2·76	No appearance of G.P.I.
	202	3·78	2·47	Very slight appearance of G.P.I.
	212	4·38	2·50	No appearance of G.P.I.
	236	3·70	3·30	,, ,,
	232	3·54	2·42	,, ,,
G.P.I. (deposit)	188	5·25	3·84	Frontal region. Well-marked appearance of G.P.I.
	189	5·50	4·38	Ditto.
	194	6·11	5·33	Deposit not in capillaries. No appearance of G.P.I.
	216	4·28	4·24	Very slight appearance of G.P.I.
	224	5·91	4·45	Typical appearance of G.P.I.

Table IV shows that there is less iron in the brains of children than in those of adults, and that normal figures are reached at 11 years. The iron content of children's blood is approximately the same as that of adults (17).

TABLE IV.—*Iron in the Brains of Children.*

		H_2O%.	Total Fe.	"Available" Fe wet basis.	Total Fe dry basis.
Newborn:	Cerebrum	87·4	0·90	0·81	..
4 days:	,,	88·7	0·80	0·42	..
8 months:	Cortex	85·8	1·80	1·27	..
	Corpus striatum	79·7	2·70	..	..
	Pallidus	76·8	7·37	..	..
2 years:	Cortex	84·1	1·34	..	..
	Corpus striatum	82·4	1·83	..	..
4 ,,	Cortex	..	..	..	12·9
	Pallidus	..	..	..	23·2
6 ,,	Cortex	..	..	..	14·1
11 ,,	,,	..	..	..	18·2
	Corpus striatum	..	..	..	76·5

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In Table V are given the results obtained when the tissue is in contact with the reagents for 30 minutes as advised by Tompsett (11). This iron, referred to by Tompsett as "inorganic", appears in excess in the corpus striatum, but not in those G.P.I. cortexes showing a histochemical deposit.

TABLE V.—*Distribution of "Inorganic" Iron in the Human Brain and G.P.I. Cortex (mgrm./100 grm. fresh tissue).*

		Fe.	n.	σ.
"Normal":	Cortex	1·28	10	0·32
	White matter	1·23	9	0·27
	Cerebellar cortex	1·39	8	0·38
All cases:	Corpus striatum	3·21	13	0·84
	Thalamus	2·15	4	0·36
2 years:	Corpus striatum	0·16	..	..
4 „	Cortex	0·69	..	..
G.P.I. no deposit:	Cortex	1·19	4	0·22
„ deposit:	„	1·29	5	0·44

Estimation of the total iron in sciatic nerve and in the spinal cord gave the following results (mgrm./100 grm. dried tissue):

	Fe.	n.	σ.
Sciatic nerve	6·6	5	0·985
Spinal cord (tissue preserved in formol)	6·3	8	0·694

It will be seen that sciatic nerve gives a much smaller value for the total iron than the white matter of the corpus callosum. It would appear improbable that the medullated nerve-fibres of the peripheral nerve have a markedly different mineral constitution to the medullated fibres of the central nervous system. It is therefore possible that the chief iron-containing constituent of the central white matter is not the nerve-fibre but the neuroglia. Some probability is given to this view by the analysis of a homogeneous glioma removed from the brain. This showed a total iron value of 20·4 mgrm./100 grm. dried tissue. It is true that the tumour neuroglia cell cannot be assumed to have the same constitution as the normal supporting glia, but should this be so, these results would indicate that the iron of the nerve-fibres is very much less than analysis of cerebral white matter would indicate. In the case of the sciatic nerve, the iron value again represents that of the medullated fibre together with an unknown amount of iron containing connective tissue. A separate estimation of the iron of the connective tissue of sciatic nerve gave a value of 5·6 mgrm./100 grm. dried tissue.

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Copper.

In his review on the biological significance of copper, Elvehjem (18) states that this element is present in all living matter, and that a complete understanding of the action of copper will bring us distinctly nearer an understanding of the fundamental processes of living matter. Traces of copper (or iron) can render reduced glutathione autoxidizable (2).

Copper has been estimated quantitatively in the whole brain (8, 11, 19, 20), and in the cerebellum in a single instance (20), but not in the various parts of the brain.

I have estimated the copper according to Haddock and Evers (21) in a solution of the ash of the tissue (recovery 90·5%; error for duplicates ± 6·5%), and have obtained the results given in Table VI:

TABLE VI.—*Distribution of Copper in the Human Brain* (*mgrm.*/100 *grm. fresh tissue*).

	Cu.	n.	σ.
Cortex	0·43	11	0·126
White matter	0·29	11	0·062
Cerebellar cortex	0·40	11	0·141
Corpus striatum	0·47	8	0·132
Thalamus	0·41	1	..
Pallidus	0·60	3	0·066
Substantia nigra	1·45	3	0·316
Cerebrum (newborn)	0·18	2	0·070
,, (1 hour)	0·18	1	..
Cortex (2 years)	0·36	1	..

The copper is presumably all "available", because Tompsett (22) found that the copper may be extracted completely from tissues by trichloracetic acid. No relative excess of copper was found in the corpus striatum, such as exists in the case of iron; but the pallidus contained more copper than the cortex; and in the substantia nigra a large excess of copper was found: in this case it was necessary to combine the tissue from a large number of brains, and three such combined estimations were made. As the substantia nigra contains large amounts of melanin, the excess copper may have some relation to the catalytic influence of copper on the oxidation of 1–3 : dihydroxyphenyl-alanine ("dopa") to melanin (19). The ink-sac of the octopus, the ink of which contains melanin, has a very large copper content (19). The substantia nigra of the ox brain, which does not contain melanin, has about half as much copper as the substantia nigra of the human brain. The copper content of the cortex was normal at 2 years.

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The distribution of copper in the ox brain is given in Table VII for comparison with human brain. The ox cortex and cerebellar cortex contained nearly twice as much copper as the corresponding parts of the human brain. [The ox liver, particularly in the newborn, contains very much more copper than that of the human liver (19). The copper content of ox blood is not higher than that of the adult (23).] The substantia nigra from ten brains gave enough tissue for a single estimation.

TABLE VII.—*Distribution of Copper in the Ox Brain* (*mgrm./100 grm. fresh tissue*).

	Cu.	n.	σ.
Cortex	0·77	6	0·171
White matter	0·23	5	0·182
Cerebellar cortex	0·77	4	0·292
Corpus striatum	0·35	5	0·123
Substantia nigra	0·75	1	..

Manganese.

Manganese is a regular constituent of animal tissues, in which it has important metabolic functions (24). Animals treated with manganese compounds show degenerative changes in the central nervous system (24).

This element has been estimated in the human brain by Reiman and Minot (25), who obtained values of 0·029, 0·024, 0·032 mgrm./100 grm. fresh tissue for a baby, a child of 4 years, and an adult (anæmia) respectively.

Estimations of the manganese content in the various parts of the human brain gave the results set out in Table VIII:

TABLE VIII.—*Distribution of Manganese in the Human Brain* (*mgrm./100 grm. fresh tissue*).

		Mu.	n.	σ.
Adults:	Cortex	0·019	13	0·0052
	White matter	0·031	13	0·0106
	Cerebellar cortex	0·023	6	0·0029
	Corpus striatum	0·053	3	0·0025
Newborn:	Cerebrum	0·020	1	..
1 hour:	,,	0·025	1	..
2 years:	,,	0·017	1	..

Owing to the comparatively low content, it was necessary to take at least 8 grm. dried tissue for each estimation. For this reason only certain portions of the nervous system could be examined. In the case of the corpus striatum

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it was necessary to combine the tissue from several brains. Three such combined estimations were made.

The method employed was that of Willard and Greathouse (26), as applied to biological material by Skinner and Peterson (27). Recovery, 103%. Error for duplicates, $\pm$ 7·2%.

It will be seen from the table that the corpus striatum contains more manganese than any other part; and that the content of the brain at birth, 1 hour, and at 2 years, is not much less than that of the adult.

Summary.

(1) Figures are given for the distribution of iron (total, "available", and "inorganic"), copper, and manganese in the various parts of the human brain.

(2) Iron (all forms) and copper are, on a wet basis, approximately evenly distributed throughout the cortex, white matter, and cerebellar cortex.

(3) A large excess of iron (all forms) and manganese, but not of copper, was noted in the corpus striatum.

(4) The pallidus and substantia nigra were found to have the highest iron content (total and "available").

(5) A large excess of copper was found in the substantia nigra.

(6) In the G.P.I. cortex, cases in which a deposit of iron was observed histochemically gave values for total and "available" iron that were significantly higher than values obtained for cases in which no deposit was observed.

(7) The typical appearances of G.P.I. were found to be associated with the histochemical deposit of iron, and so also with the high figures for total and "available" iron.

(8) Less iron (all forms) was found in the brains of children of six years and under than in those of adults (normal at 11 years). At two years copper and manganese were approximately normal. [In the livers of children, iron, copper and manganese fall during nursing and increase during childhood (28).]

(9) In the newborn, iron and copper are low and manganese normal.

(10) The ox cerebral and cerebellar cortex were found to contain more copper than the corresponding parts of the human brain.

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AN UNDECIDED COMPENSATION CLAIM ARISING FROM THE SUICIDE OF A VOLUNTARY PATIENT.*

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A. THE FACTS OF THE CASE. BY DR. DRURY.

THE case reported here concerns the suicide of a voluntary patient and a subsequent claim for damages for "negligence, breach of contract and warranty", which, after many delays, was struck out of the Assize List on account of the non-appearance of the plaintiff.

The facts are as follows: On September 3, 1935, a doctor telephoned the Medical Superintendent of the hospital, asking him to take a patient of his, A. V—, who desired to come in as a voluntary case. This was agreed to, and the patient was admitted the same day, having signed a request form incorporating the requirements of sect. 1, sub-sect. 1 and 5 of the Mental Treatment Act. On admission he was found to be depressed and worried, complaining that he was a failure, etc. No definite delusions could be ascertained, and he had good insight. Though there were senile changes present, he was in quite good health and condition for a man aged 66, who had spent a sedentary life as a clerk until his retirement on pension some six years previously. His history showed that he had once been a patient at Park Prewett with a similar mental condition. He had also on many occasions spoken of suicide and had some years before made two or three crude but not serious attempts.

At first he was kept under constant supervision, but as he improved he was given a certain measure of parole—i.e., to go alone to the canteen and walk in the ward garden, and later still to go out on the cricket ground with others not under special supervision. During the time he was in the hospital, September 3 to October 31, he improved, became much less worried, and though he was never a bright or cheery person, he appeared towards the end of October to have practically reached his normal state.

On the morning of October 31 he was in the ward garden, where he had been seen and spoken to at 11.0 o'clock. Fifteen minutes later, when called

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in for a shave, he was found missing. This garden is surrounded by a 6-ft. railing, screened by shrubs and small trees. Apparently he had climbed the railings—no mean feat for a man of his size and age—and walked past the farm to the main road. Here he was run over and killed by a light lorry. The lorry driver was the only witness of the affair, the road being empty of traffic at the time of the occurrence. He stated at the inquest that A. V— was on the opposite side from the hospital gate, and that as he approached, A. V— attempted to run across the road. Personally I am inclined to believe that A. V— became agitated and suddenly tried to get back to the hospital. However, as he was a patient in a mental hospital, the verdict was almost of necessity one of " suicide whilst of unsound mind ".

Mr. Freer, Solicitor and Clerk to the Committee, deals more at length with the legal aspect of such cases. To my mind the salient points are :

1. The Plaintiff would have had to prove negligence on an application to the High Court before he could bring an action at all.

2. He would have had to prove that the present system of graduated parole was wrong, and give a better one.

3. That even from prison, escapes take place.

B. Legal Aspects. By Mr. Freer.

I need not go into the general principles of the law of negligence so far as doctors and other professional men are concerned, for I have no doubt you all know that the basic principle is that a doctor or other professional man is guilty of negligence only if he does not use that degree of skill which is expected of practitioners skilled in medicine, or (e.g.) lawyers trained to the law.

They are not to be presumed negligent because they have not shown extraordinary skill. If, however, they have not displayed that degree of skill which is expected of them they will be guilty of negligence, and damages will follow in the absence of contributory negligence on the part of those treated or advised. Negligence is well described as " the omission to do something which a reasonable man guided by the considerations which ordinarily regulate the conduct of human affairs would do, or doing something which a prudent and reasonable man would not do ".

We are going to deal particularly with the recent case of A. V—, who was accepted into this hospital as a voluntary patient, and who subsequently escaped and was killed by a lorry outside the hospital grounds. The Medical Superintendent and his assistant both agreed that A. V— on admission had volition and knowledge of his surroundings and general knowledge of what he was doing, and he was thereupon accepted as a voluntary patient, and signed an undertaking not to leave the hospital without 72 hours' notice. I may mention here that the Mental Treatment Act, 1930, s. 1 (5), stipulates that a person received as a voluntary patient may leave the hospital, etc., upon

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giving 72 hours' notice. Obviously, therefore, if he leaves without this notice the patient is in default, which would be prima facie a defence to an action.

On the other hand, the question might arise as to his ability to keep any undertaking he has given at the moment he leaves the hospital, which might be an unascertainable factor.

Therefore, it seems to me that the authorities, if involved in an action, might not be able to stand on the lack of notice, and this might prove an unreliable defence on which to lean.

The corollary to this appears to me to be that there is no virtue in taking a written undertaking from any voluntary patient, having regard to the fact that one must assume that all of them on entering the hospital are abnormal. On that point I think the authorities might be well advised to consider the following alternatives :

(1) If they wish to adhere to the undertaking which is used at the Narborough Mental Hospital, then I think this ought to be fortified by an indemnity from the man's wife or other close relative, and this having regard to the fact that an action for negligence would survive to the man's next of kin provided damage had been sustained.

In this particular case, if the authorities had been guilty of negligence, damages would flow on account of the fact that the man was in receipt of a pension, and his representative would set up that his expectation of life might have been several years. This claim must be considered always to arise in the case of a man who was or may be a potential bread-winner.

(2) Obtain a definite statutory indemnity in the case of a mishap of this sort, and this case calls special attention to the risks the authorities must run in treating a case of this sort. This would probably be very difficult to obtain on the ground that an absolute indemnity might lead to slackness, and therefore the first alternative is preferable if practicable.

In this connection, we must consider for a moment s. 16 (1), (2), (3) and (4) of the Mental Treatment Act, 1930. That section with its sub-sections is obviously meant and intended to give a sufficient indemnity to hospital authorities. In my opinion it certainly does not do so. What it says in fact is this :

S. 16 states that s. 330 of the Lunacy Act, 1890, and s. 63 of the Mental Deficiency Act, 1913, shall take effect as if the following (viz., s. 16, s.s. (1)) were substituted for s. 330 of the Lunacy Act :

" 16 (1) Where a person has presented a petition, etc. . . . *or has done anything* in pursuance of this Act (i.e., Lunacy Act or Mental Deficiency Act) *or any Act amending this Act* (i.e., the Mental Treatment Act, 1930), he shall not be liable to any civil or criminal proceedings whether on the ground of want of jurisdiction or on any other ground unless he acted in bad faith or without reasonable care."

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This means, as I read it, that a person who has done anything under the Mental Treatment Act, 1930, shall not be liable to proceedings unless he acted without reasonable care. If this is so, under s. 16 (2) no proceedings can be brought without the leave of the High Court.

On these two points I have the following remarks to make :

I do not think this forms a perfect indemnity, because grounds of action of the type we are considering must be always found on an allegation that the authorities have not used reasonable care. By s. 16 (2) the opinion of the Court has to be invoked and leave obtained to bring the action.

It would appear therefore that the leave of the Court would be obtained after the Judge had heard the evidence, and he would then declare whether or no there was a case to go to the jury.

I have no doubt that s. 16 (1) clearly applies to the Mental Treatment Act, 1930, and that therefore this action could not have proceeded until the Judge had certified there was a prima facie case of negligence.

Dr. Drury has detailed to you the steps which were taken in order to safeguard this man and to assist his recovery, and I have no doubt at all that the most adequate care was taken by the authorities, and that what has happened, although unfortunate, is impossible to avoid from time to time if the system which is approved by psychiatrists is to be effectively tried out. Dr. Drury will say, in spite of this happening, that the system is sound, and on the whole successful in practice. I am sure you will all agree with him that the system on which the treatment of this man was based is far superior in practice to the alternative of closer confinement and watch. At the same time, I think we must also agree that this system, if it is to be worked at all, must involve in suicidal or homicidal cases risks which are bound to arise, and may at any time involve the authorities in an action of this sort.

Of course I need not say that the authorities have the fullest backing and encouragement of the Board of Control.

On the question generally of the treatment of these patients, I have neither the ability nor the knowledge to dilate upon or criticize the principles employed, but as I understand it the method of treatment is to give increasing liberty to all patients showing improvement, and I understand this treatment has had a large measure of success, inasmuch as it tends to give confidence to patients and therefore helps in the process of cure.

It is a very interesting thing to me to hear what a large proportion of patients are in fact on parole.

I can appreciate that in the case of patients who are not suffering from suicidal or homicidal tendencies, but it is obvious that a greater risk must necessarily be run if the system is applied to patients in whom these tendencies have been present, and that such cases clearly require very great and watchful care and judgment.

We will assume therefore that in this case the utmost care and judgment

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has been used, and that (within the system employed) every reasonable safeguard has been taken to prevent escape and mishap. I have, however, some hesitation in making up my mind whether the authorities might be protected where the treatment has failed and the result is death, and whether conceivably on an application being made for leave to bring an action, the High Court might not say, " In spite of your system and theories, the fact remains that this man escaped, with fatal consequences, and therefore, in our opinion, there is a prima facie case for the hospital to answer ". From that, assuming the action had proceeded and the case had come before a jury, I think it might be very difficult for a body of men without the fullest understanding of the principles and reasons for the increasing liberty given in such cases to see anything beyond the fact that the basic principle employed failed in effect, and in fact failed so far as a layman (apart from an expert) was able to see. They might conceivably go further than this, and make up their minds that there was evidence of negligence and base it on these grounds, viz. :

That the liberty recommended by psychiatrists tends towards the possibility of the grant of premature freedom before the destructive instincts of the patients have been eliminated, and they might conclude that in this case there is ground for deciding that the system (on account of its urgency) has not warranted or produced the care which was needed.

It seems to me that there might be some difficulty in persuading a jury that the system is effective, and once they came to that point of view, they would be bound to apply it (and possibly might have done so in this particular case).

It is on these grounds I have doubts as to the verdict, and this as I have said on account of the incalculable human element of uncertainty and latent unreliability which is bound to be risked if the principles followed are to be successfully applied and acted upon.

There remains another defence which is always open in the case of a claim for negligence and that is the question of " contributory negligence ", but I think that this cannot be pursued having regard to the state of mind the man was in on admission, and is bound to be in at the time he burns his boats and escapes with the consequences in this particular case.

It may be of interest to you to follow for a moment the process of this case so far as it went :

1. The action was launched within the time provided by the Public Authorities Protection Act.

2. The action was proceeding normally, viz., (*a*) statement of claim ; (*b*) defence ; (*c*) directions as to trial, etc.

Meanwhile the patient's widow had gone to America and remained there and thus the action missed two assizes. The widow made no attempt to return to England, and her lawyers had great difficulty in ascertaining her whereabouts from time to time. Finally an application was made on behalf

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of my Committee for the action to be struck out on the grounds of want of prosecution, and on the hearing of this application the Registrar ordered that unless the case was proceeded with diligently within 21 days and £25 deposited towards the costs, the matter was to be struck out.

In fact, the action was not proceeded with and the deposit was not paid and therefore the action was automatically struck out.

With regard to the costs, it is a very important thing to avoid such actions, because even if in the result the authorities were successful, they quite possibly would not be able to look to the defendants for payment of their costs.

I venture to hope that my remarks will furnish sufficient grounds for an interesting discussion.

Discussion.

Dr. DRURY: I may mention that I was the only person to whom the deceased expressed any suicidal tendencies at all, and that was during a very long conversation; during that talk he said he was fed up with life. He had carried out two or three perfunctory actions, such as tying a handkerchief round his neck.

The PRESIDENT: Was he a private patient, and is there any difference in the matter of responsibility between a private patient and a patient accepted as a voluntary rate-aided one?

Mr. FREER: That point was raised at one time. I think that at one time the Board of Control said that if they were to give evidence it would be to say that, in their opinion, there was no difference whatever. It is a legal matter, but the Board of Control would take that view.

Dr. R. M. STEWART: I do not know against whom the claim for damages was made; was it the medical superintendent, or the visiting committee? The London County Council make the medical officer shoulder all the responsibility.

The PRESIDENT: And the question in law is as to whether the committee have power to accept responsibility.

Mr. FREER: There is a Section in the Lunacy Act which says any action brought against a visiting committee may stand and run in the name of the clerk. It does not say, of course, that the clerk must pay; the visiting committee must foot the bill. The action was brought against the clerk, but he has an indemnity against the visiting committee; in fact the visiting committee are the responsible persons to defend the action and foot the bill.

The PRESIDENT: If the action was brought against the medical superintendent, have the committee the power or the right to defend it?

Mr. FREER: I would like notice of that question. It was fully discussed in a recent action, which you may remember, against the Hampshire County Council, where the Duke of Wellington was Chairman. The action was brought against the Duke of Wellington and the medical committee, and the Judge struck most of them out, leaving the clerk in at the end. He must be the nominal defendant, and in any case the visiting committee would be responsible, even if an action was brought against the medical superintendent.

The PRESIDENT: As Dr. Stewart just said, the London County Council claimed that they had not the power to defend it, and that the action must be brought against the medical superintendent. In one case, at Hanwell, it was left to the medical superintendent.

Dr. HOPKINS: It was left to Dr. Litteljohn to defend.

The PRESIDENT: And my committee insisted that all the medical officers should be members of one of the Medical Defence organizations.

Mr. FREER: I do not think that has ever been decided, and therefore in cases against mental hospitals it is the practice, in the legal profession, to put up as many people as possible, and see what happens.

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A MEMBER: Must application be made to the Judge in Chambers before a case comes forward?

Mr. FREER: I think that is what would happen. In this case the action was never set down; but an application would be made to the Judge in Chambers.

A MEMBER: I ask for further consideration of the wording of the title of the communication, "Suicide of a voluntary patient". Would not the relatives have to prove that it was suicide before they could bring an action? It seems likely that it was a case of accidental death, and if it were so, how would that affect the position of the defendants in a possible action?

The PRESIDENT: Ought the patient to be in a position where he could meet with an accidental death of that sort?

Mr. FREER: The coroner returned a verdict of suicide.

A MEMBER: Surely that means nothing?

Mr. FREER: We were faced with a stiff case when it was said to be suicide, and the only witness who was present said the patient ran in front of the lorry.

Dr. PETRIE: When a jury has whitewashed the patient it did not invalidate subsequent findings. Would you advise that you can bring back a voluntary patient who has escaped? Possibly you could if you had reason to think he was about to commit an offence against the law. This man was at large; would you have recommended that the officers should re-take this patient, apart from whether they thought he was about to commit an offence against the law? Was not the contract broken off because he escaped from the institution?

Mr. FREER: A coroner's jury does not bind the High Court, and the case would eventually have been tried.

Dr. MASEFIELD: Would it have made any difference if 24 hours before he escaped he had signed a form of notice of his intention to leave the hospital? Would that have made any difference to the proceedings afterwards, within 72 hours?

Mr. FREER: I do not think it would have made any difference if he had not allowed the 72 hours' notice to expire. In answer to Dr. Petrie, he had not given the 72 hours' notice, and therefore he was under contract to stay in the hospital. But there is the point I raised in my paper as to whether the notice is any good at all.

Dr. PETRIE: As he had left the hospital he had broken the contract and was at liberty to do as he chose?

Mr. FREER: I do not think we could take that line, because he escaped when he should not have done so.

Dr. PETRIE: Some of us have been advised that if we re-take a non-suicidal voluntary patient we shall be imperilling the means which can be taken by the Medical Defence Union.

Mr. FREER: I should like to see the arguments on that, because if he goes without giving notice he breaks the contract and he is liable to be re-taken.

Dr. PETRIE: You advise sending an escort and bringing the patient back by force even if he has said, "I do not want to enter the hospital"?

Mr. FREER: I should see what state of mind he was in. If he was normal one would have the right to go against him or his representatives for breach of contract.

The PRESIDENT: Is not arrest a breach of contract?

Mr. FREER: It depends on his state of mind.

Dr. FITZGERALD: On a question of law of contract you cannot detain persons under such conditions; you are open to an action for damages if you restrain a patient; you can only pursue by civil process. How can you arrest or detain a voluntary patient who wishes to leave? In law you are committing an assault if you do.

A MEMBER: It has happened to me, and it was said we must not send for the patient. We concluded we had no right to bring him back.

Mr. FREER: I am talking now without the book, and I am very interested in what you say, and if counsel advises you otherwise I shall not say he is wrong. It is an interesting point.

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A MEMBER: We might safeguard ourselves by getting the relatives to grant us an indemnity against such a happening. If so, what form should it take?

Mr. FREER: A form of indemnity is an easy thing do draw up; it would be a statement that the patient is in the hospital for treatment, and if an unfortunate accident should happen to him, the representatives will not bring an action for damages.

The PRESIDENT: That would delay and interfere with having voluntary patients.

Dr. DRURY: One's contract is with the voluntary patient; I have nothing to do with the relatives; my contract is with the patient who asks me to take him in for treatment.

Mr. FREER: That is so, but still, after a patient is dead you must deal with his relatives.

A MEMBER: We are told by the Board of Control that the Act does not say the patient cannot leave without notice; therefore he has a right to leave, and if he does leave, what right have you to bring him back?

Dr. MASEFIELD: You believe him to be a person of volition, and that he wishes to leave, but he has not fulfilled the requirement of 72 hours' notice. But if he has given notice of his intention and shows his desire to leave, Dr. Drury would be a bold man—even without any accident—if he sent to have him taken back.

A MEMBER: A voluntary patient says, "I am leaving the ward", and attacks the nurse and takes the keys; has the nurse the right to hold him down and take the keys away from him? The patient says, "I am entitled to take the keys to let myself out".

Mr. FREER: If he attacks people in the hospital, it is their duty not to allow him to go out.

A MEMBER: This is an important discussion; can we have a show of hands from the medical superintendents present on the question whether we send for patients who have escaped from the hospital?

Dr. PETRIE: You can notify the police, so that he receives attention at their hands.

A MEMBER: I have sent for the police and got a patient back many times. On one occasion a patient was found drowned in a canal, but he had left the premises.

The PRESIDENT: A voluntary patient may lose volition for a month and still be retained as a voluntary patient, and he may need seclusion; I have secluded several voluntary patients. The medical officer is responsible for preventing a patient from going out.

Dr. MCRAE: There is no object in a man coming to your institution and in agreeing to give three days' notice if you have no power over him during those three days. You can treat a voluntary patient exactly as you treat certified patients—that is, for three days; otherwise the voluntary patient system breaks down. The wife of a patient of mine owned a public-house, and she said to the patient, "You are not going home with me; you sign that paper saying you will enter the hospital." He agreed. That evening he had delirium tremens and was climbing up the wall of the padded room. He was secluded for three weeks. He got better. But before I allowed him to go I had him in the office and said to him "If you are vindictive you may turn round and say I had isolated you and detained you against your will, and I ask you, Will you do anything like that? I locked you in the padded room; did I do right?" He said, "You did right; I will say nothing about it". I may say he was going home to the same wife!

The PRESIDENT: When we gave evidence before the Royal Commission Lord Macmillan doubted whether such a patient could be a voluntary patient. Yet that Commission recommended that voluntary patients should be allowed. They can remain for one month after losing volition. It all points to this—that you must give them any necessary treatment during that time. If those patients are likely to damage themselves you are bound to restrain them. I had one case,

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that of a man on a bicycle who ran into a bus and broke some ribs, and the only regret of the relatives was that the accident had not been fatal. You are bound to protect the patient.

A MEMBER: There is a difference between what you will do when a patient is in your institution and what he will do when he escapes.

The PRESIDENT: It depends upon the mental state of the patient, and you are responsible for deciding that.

Dr. PETRIE: When you take a patient back, unless he resigned the form of contract, you treat him, as Dr. McRae said, in the way you think fit.

The PRESIDENT: If a patient is considered to be unfit I would not care to take the responsibility of declining to take him back, and that is the line I intended working upon.

Dr. MCRAE: Could we not take the line which Dr. Saxty Good expressed to me, that nobody knows a patient is suicidal until he has committed the act. It is a fastidious attitude of the law to expect us to know what a person will do; even the law cannot make us know what we do not know.

A MEMBER: I ask whether the request a voluntary patient makes is really a contract. It is a request to be treated and a promise to obey the rules of the hospital while he is undergoing that treatment.

Mr. FREER: There is no statutory form, but it is parallel to the case when you go into a hotel; it is a contract to the effect that when you pay the money you receive certain benefits, and you are entitled to those benefits.

A MEMBER: A patient of mine wanted to sell a number of shares, and said he could make £50 out of the transaction. I disallowed him to do it, but he could have made that sum. Am I liable for his losing the amount? Could he have brought an action against me for it?

Dr. DUNCAN: I had a case which was practically identical with the circumstances of the case we are discussing, two years ago, when a patient at Severalls made an unauthorized departure one morning without giving notice, and, the Medical Superintendent being absent, I told the clerk to the hospital that he had gone and that I considered it was equivalent to his having left without notice, and to strike him off the books, and that was done. The next day the patient was found drowned, and was taken up on the Suffolk side. I was called upon to give evidence as to his mental state, and I did so, saying that he had been a voluntary patient at the hospital, but was no longer my patient, and I presumed it was correct. Therefore, under the circumstances, the hospital was covered against any possible action at law. And I think the same applies here, except that in the present case the patient had not been struck off the books.

A MEMBER: Is not there a case in the High Court in which a decision was given in such circumstances; that it is a criminal act and therefore the relatives cannot benefit by it?

The PRESIDENT: It is a criminal act if the person is not of unsound mind.

Mr. FREER: If he is of unsound mind he is, like a minor, not responsible.

A MEMBER: A section of the Act says a voluntary patient is not responsible for his torts.

The PRESIDENT: A person of unsound mind who is a Member of Parliament can remain a Member of it for six months. After such a Member has been under care six months, the Speaker in the case of the Commons, the Lord Chancellor in the case of the Lords, is notified. I had a member of my own committee who became a patient.

Dr. J. H. MACDONALD: All this has been very interesting, and it has raised one point which has been very prominent in the discussion. I do not know the law in its details as applied to England with regard to the admission of voluntary patients; but in Scotland, where voluntary admissions have been prevalent for many years, in some hospitals there is a tendency to overdo the admission of voluntary patients. There is no doubt in my own mind that many voluntary patients are admitted to mental hospitals who are definitely insane and certifiable: I have taken a hand in that matter myself, and I have admitted persons as voluntary

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patients against my sounder judgment. A certain number of voluntary patients are certifiable and, in my reading of the Scottish law, it should not be so. For a person to be rightly regarded as a voluntary patient he should not be in a certifiable condition. This present case which we are discussing raises the same very important matter. We overdo the admission of patients as voluntary ones and so we are stultifying the compiling of statistics, which are valuable, but at the same time are faulty and unreliable; these cases are not included in the statistics of the insane, though they are known to the Board of Control. It is the first instance I have known of a voluntary patient committing suicide, and, to me, it makes the difficulty all the greater, and I shall not, in future, have much to do with the admission of certifiable patients on a voluntary footing. Once a patient—whether voluntary or involuntary—comes to me it is my duty to protect and look after him in every way, to the end of his days if he is a voluntary patient. I am responsible for him in every way.

The PRESIDENT: Because the voluntary system has gone on without actions having been brought it was thought to be good, but people are tending to bring actions in a way they never did before.

Dr. FLEMING: Will Dr. MacDonald tell us what he means by certifiable patients?

Dr. J. H. MACDONALD: If I can certify a patient as being, after examination, of unsound mind there should be no difficulty in saying he is certifiable. When I certify a man to be of unsound mind you can take it that I feel no doubt about it.

Dr. FLEMING: Surely one of the objects of the Act is to admit certifiable patients as voluntary patients in order to avoid certification.

Dr. DRURY: A G.P.I. patient who was in a state of raging mania asked to be voluntarily admitted. He thought it was a hotel. He came and said, "I want to come in here". In five days' time he was in the middle of malaria, in a most violent and desperate state, and he gave his notice while he was like that. I said, "You are not going", and he said, "I am going". I said, "If you persist in giving your notice I shall certify you", and I did so. Afterwards he became much better, and six weeks later he was discharged. I have had two letters from him since, thanking me for what I did.

Dr. G. W. SMITH: I would like to thank Mr. Freer, on behalf of you all, for his brilliant exposition of the law. I ask whether he knows of any cast-iron indemnity against the attacks which we are liable to as members of a profession who have to do precarious things. You, Sir, have said you have dealt with voluntary patients very frankly, and have taken the law into your own hands in regard to them, and therefore I ask whether Mr. Freer knows it is the case that there is no indemnity against prosecutions or actions for damages, and that, in the end, we have to rely on the sagacity and generosity of the Judges, either in quashing cases in Chambers, or seeing that damages are not awarded against us. Does Mr. Freer know of any means by which we can indemnify ourselves against actions for damages?

Mr. FREER: No, I do not. I agree with every word Dr. Smith said. I think that, in the end, you must rely on the ability and consideration of His Majesty's Judges, and possibly on the common sense of a jury, if you have to go before one. But in the case of an indemnity I suggest it would be a help to have one, though it would not be "cast-iron". The construction of the indemnity would be the duty of the Judge, not of the jury. But I could not recommend anything which is cast-iron, any more than you gentlemen can recommend any treatment which is cast-iron.

The PRESIDENT: Do the Committee of this hospital propose to have an indemnity?

Mr. FREER: No. I am not altogether the father of this medico-legal child. I have had very great help in regard to it, and it has only just been produced, and my Committee do not know very much about what is in it at present.

The PRESIDENT: You will delay the admission of voluntary patients if you are going to wait for an indemnity.

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Dr. PETRIE: And relatives can interfere with taking patients out. It may be against the interests of the patient if you require an indemnity.

Dr. MCRAE: Does not a difficulty arise in the matter of the framing of the Act, which says they must prove that you did not exercise reasonable care? It is that adjective "reasonable" which is the snag. You are depending on His Majesty's Judges. When this Bill was being discussed in London I was going to make observations to the Committee about faults in the drafting, but a colleague from Scotland said to me, "You sit quiet; you come from Scotland; it has nothing to do with us". The Bill is as far as Parliament would go.

At the conclusion of the discussion a vote of thanks to the readers of the paper was carried by acclamation.

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SYPHILIS IN MENTAL HOSPITAL PRACTICE.*

By DAVID PRENTICE, M.B., Ch.B., D.P.M.,
Deputy Medical Superintendent, Narborough Hospital.

Dr. PRENTICE said that syphilis, in its incidence, diagnosis and treatment, presented, in mental hospital practice, problems which differed from those met with in dealing with the disease in general practice and in V.D. clinics. In the former its incidence was higher than in the general population, and that was largely because many of those whose nervous system had become affected by the later stages of the infection ultimately developed a psychosis. Drugs which were efficacious in somatic syphilis showed but little therapeutic effect in the treatment of the nervous system when affected by syphilis. There was a wide variation in the syphilis occurrence-rate among new admissions to mental hospitals, namely, from 5% to 31%; there was no doubt that incidence varied in different parts of the country; for instance, at Whittingham Mental Hospital, Lancashire, the male admissions in one year showed 21·9% with syphilis, and females 8·9%. At Narborough in the past two years—using the same methods of diagnosis—the positive males were only 7·7%, the females 4·6%. It was difficult to estimate reliably what proportion of the general population suffer from syphilis, but comparison of the figures of the Royal Commission on Venereal Diseases with those given by numerous workers in mental hospitals—excluding cases of general paresis and meningo-vascular syphilis—showed that the part played by syphilis in the ætiology of ordinary mental diseases must be a very small one. Bearing in mind the body-mind relationship, any toxic or infective process which could be a factor in the ætiology of mental illness should be dealt with. Even if the disease were predominantly psychogenic, all possible physiogenic factors should be eliminated or dealt with.

In view of the lack of inhibition and the disorders of conduct characterizing the preliminary stages of some mental diseases, it was surprising that the early stages of syphilis were not more often encountered. During ten years' examination of recent admissions Dr. Prentice had never seen a primary sore, and it was rare for him to see the ordinary clinical signs of the secondary and tertiary stages. All the cases diagnosed as positive before the routine test was inaugurated suffered from either general paralysis or meningo-vascular syphilis; no cases of latent syphilis were detected. The marked increase of diagnostic efficiency constituted a strong plea for the more general adoption of a routine serological examination.

The Wassermann test had acquired a prestige and authority which on the evidence now available was not wholly justified. No present biological method of examination had reached mathematical accuracy in regard either to sensitivity or specificity, but the general opinion among serologists at present was that the best of the precipitation tests had been proved to be superior in regard to sensitiveness, and equal with respect to specificity, to the Bordet-Wassermann reaction and its modifications. Most of the flocculation tests gave good results in the hands of their originators and experienced workers, and their simplicity made them eminently suitable for carrying out at ordinary mental hospital laboratories.

* A discussion held at the Quarterly Meeting of the Royal Medico-Psychological Association, May 21, 1937.

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Meinicke recently concluded (1) that the flocculation and the Wassermann test, had now reached the apex of their efficiency, and could not be further refined without losing in accuracy; (2) that where differences in precipitation in the various reactions or in different modifications of the same reaction occurred, these were only quantitative and therefore relative, even if the difference appeared to be absolute in the weaker methods; (3) the practitioner of the future must make use of the modern sharp flocculation reactions for correct diagnosis and for the control of therapeutic effect.

To avoid possible error it was generally advised that two separate diagnostic methods should be employed simultaneously. The micro method offered several advantages in mental hospital work, especially when dealing with troublesome and resistive patients. In its latest form (M.K.R. II) the Meinicke reaction could be recommended as simple in technique and reliable in results; moreover the readings could be made on the same day. The ideal method was to do two or more different tests on each serum.

Cases of somatic syphilis could be treated with the usual arsenicals and bismuth and mercury, though they did not appreciably alter the course of syphilis of the nervous system. The one drug which had given good results in treatment was tryparsamide; it was a valuable aid in arresting the infection. For best results the treatment should be prolonged over several courses; there was only slight risk of damaging the vision as a result of its use. It should be stopped at the first sign of visual deterioration.

A therapy for which good results were claimed was the intramuscular injection of cerebral lipoids: 1 c.c. was injected every other day, with a weekly injection of tryparsamide. The remission-rate following this was high: patients gained in weight, and the basal metabolic-rate returned to normal.

The mode of action of the fever-producing therapies had not yet been adequately explained; there might be a direct bactericidal effect on the organism, or a stimulation of the general metabolism, with resulting special tissue and biochemical changes. A specific antagonism between the two organisms was supposed to exist under malaria therapy. In some cases this latter had proved superior to any form of drug therapy.

Physical agents included hot baths, the electric cabinet, and short waves or diathermy; the main mechanism was the heat production.

Of chemical agents, sulphurated oil and protein shock preparations were examples. He had treated 25 patients by means of sulphurated oil, but the percentage of remissions was not sufficiently high to warrant it being continued, and malarial treatment was resumed.

A third group consisted of the injection of preparations of dead, or attenuated live micro-organisms, examples of which were pyrifer and T.A.B. vaccine.

Of the infections, benign tertian malaria remained predominant, and had stood the test of twenty years of continuous use. Occasionally a patient proved immune to inoculation, and the spacing of rigors could not be so controlled as by other methods of treatment. The serious malarial illness was often followed by anæmia and debility. Yet it remained the chief therapeutic agent, and represented the greatest advance yet made in the treatment of syphilis of the nervous system.

Discussion.

Dr. FORD ROBERTSON said he would like to make a few remarks on the diagnostic value of the flocculation tests in psychiatric practice. There was no doubt that these were recognized as possessing a sensitivity which was much superior to that of the Wassermann test. In the past three years he had made a routine practice of running three different Wassermann methods at the Institute—the standard method for psychiatry, the Wyler drop method, and the Browning method—with three flocculations—the Kahn, the Meinicke, and the Müller reactions. There had been more agreement with regard to the sensitivity of the three flocculation tests

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than among the Wassermanns. The flocculation tests had a much higher sensitivity. The difficulty in regard to flocculation reactions in mental hospitals was that none except the Kahn was as reliable as the Wassermann for fluids; even the Meinicke failed to some extent. It was agreed that the Wassermann was still the test *par excellence* for fluids. The difficulties in regard to fluids had been so marked that in the past two years he and his colleagues had tried to solve some of the problems in these electro-colloidal reactions known as flocculation tests, and he felt that some of the faults were now being overcome. He hoped that in the next few months the Institute would be able to produce a flocculation test involving some of Meinicke's principles which would have equal sensitivity and specificity for fluids and sera, and which, therefore, could be adopted in psychiatric practice.

Dr. E. J. FITZGERALD said that this was a subject in which he had been interested for years, and he could not agree with some of the conclusions which were set out in the paper concerning the Meinicke reactions. He felt no doubt, from a long experience, that the Kahn was superior to other reactions in respect of blood and cerebro-spinal fluid; especially was it superior in the blood. The Kahn had the great advantage that it could be carried out by the average medical officer who did laboratory work. The Meinicke he regarded as very inferior to the Kahn, but there was the advantage that a nurse could collect the blood. A great disadvantage was the unduly large number of false positives. It was necessary to have two tests, the first a diagnostic one, and the second an exclusion test. One test gave the maximum sensitivity; the other must give absolute specificity, while not troubling one so much about sensitivity. In the diagnosis of syphilis, active and latent, there was no test which could compare with the Kahn. It was absolutely necessary to have a routine examination of both the blood and the cerebro-spinal fluid. Dr. Nicole and he were struck by cases in which one got a negative report for blood and a positive one for cerebro-spinal fluid. He sent a sample of blood, taken on the same day, to three different laboratories, and got one weak positive, one doubtful, and one negative. A week afterwards he sent the same sample and got opposite reports from the same laboratories. In the hands of even competent people, the Wassermann reaction alone was a most fallible method of diagnosing syphilis available, and the League of Nations had pointed out, and it was supported by certain well-known syphilologists, that to diagnose syphilis on the Wassermann alone amounted to malpraxis. Most laboratories would return a report of both Wassermann and Kahn.

With regard to diagnosis by the Meinicke, for five years he routinely examined the blood, dealing with over 200 admissions a year. On an average, 21% of the males admitted had syphilis, but he was struck by the fact that in very few did syphilis seem to have had anything to do with the mental illness, apart from the general paralysis group. Dr. Shaw Bolton had postulated that if a man had a weak brain he would break down at a certain age, and if he had syphilis, he would get general paralysis. Excluding general paralysis, the percentage of cases of syphilis in mental hospitals was much below those in the general population.

He would like to pay a tribute to Dr. Prentice, because that gentleman had introduced him to the study of serology.

He wished to emphasize that it was incorrect to say that the various methods possessed anything like the same value. The League of Nations' reports showed that the routine Kahn was infinitely superior to the best Wassermann.

Dr. DUNCAN said that the present was the first occasion on which he had heard it advocated that nurses should take from patients blood for serological tests. Against such a practice he wished to enter his protest. For whatever purpose, the taking of blood from patients should be done only by the medical officer. It was the medical officer's duty to take all possible precautions.

He had listened to Dr. Prentice's paper with the greatest interest, and agreed with everything that gentleman had said. As to results of the Wassermann test, he mistrusted every laboratory except his own.

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Dr. R. M. STEWART said that Dr. Prentice had mentioned an extraordinarily high percentage of male admissions who had syphilis—he thought it was 21%—and he asked to what years those figures applied. The newer forms of therapeutic agents had now been used for so long that a substantial reduction in the incidence of syphilis should be seen. Credit for such reduction must be given to the education of the public and to their increasing confidence in treatment, rather than to the potency of the therapeutic agents in the arsenical group. In Vienna all forms of syphilis were now being treated with malaria. He thought that example would have to be followed in this country. If he were in charge of a large acute mental hospital he would give every patient who had presumed active syphilis a course of malarial treatment.

The PRESIDENT agreed that 21% seemed an enormously high figure.

A MEMBER said he had been struck by the definite fall in the incidence of syphilis in the last ten years. At a certain hospital 19% of the admissions in 1926 gave a positive reaction, and there were 21 cases of general paralysis of the insane, whereas in 1936 the incidence-rate for syphilis was 6·3%, and there were only 4 cases of general paralysis.

The PRESIDENT said his own experience, too, had been that the incidence of cases of general paralysis of the insane had fallen a great deal in recent years. He was trying to collect the incidence figures for that disease over a course of ten years. Apart from the places and institutions where there were many sailors, there could be no doubt about the fall.

Dr. DRURY said that when he started at Warwick Mental Hospital there were 25 cases of general paralysis, half of them ambulatory. At the present time, with a similar population, there was only one case of that disease.

Dr. DONALD ROSS regretted that, with this discussion in prospect, he had not looked up his figures bearing upon the subject. Some might remember that fourteen years ago Dr. Ferguson Watson contributed to the Journal an article giving an analysis of a series of examinations of serum in various mental hospitals. At that time in his own institution there were 415 cases, and more than 400 of them were tested; it was all controlled, and no data were supplied to the examiners. Two or three samples from patients went under different numbers. The staff, on their side, took careful notes, so it could not be alleged afterwards that the clinical findings had been "cooked". Three flocculation tests were used. Out of 410 samples there were 25 positives. An interesting thing to him, the speaker, then was that in every case in which the clinician knew there were positive findings from elsewhere, or definite physical signs of the disease, the return was positive, and in no others, or practically none. Among the 3 or 4 exceptions was an old man who had been in the institution several times. In the ensuing 18 months he had a Charcot joint, and he died with syphilitic manifestations. Amongst the other cases which interested him at that time were two of the oldest inhabitants, a male and a female, who gave markedly positive results. The man had been admitted in 1869, and the woman in 1872. At about that date there were in the institution very few instances of syphilis, except cases admitted from Glasgow.

Last year two workers in Edinburgh were very enthusiastic about monkey malaria. The speaker had 5 recent cases which were injected with serum. They developed slight pyrexia, which lasted 5 days; none of the other cases gave any response. One gave no reaction but improved enormously.

Dr. POOL said he had been surprised to note that speakers who particularly stressed serological reactions did not mention the Sigma test. That was one of the earliest, and also one of the best, as it expressed the syphilitic infection in

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terms of units; and when one was following one's cases through, and having the blood tested at intervals, one could watch progress either way in units. Where the tests were done by one person it was a very useful test, and compared favourably with the other flocculation tests which he, the speaker, had handled.

With regard to therapy, he had had experience of the inductotherm during the past few months, and was very pleased with it. With it one could guarantee the temperatures, and it did not upset the patient in the same way that malarial rigors did. One could give any number of rigors up to 20, the average period for the development of each rise of temperature to 105° being 2½ hours. At his institution there had been 34 cases so treated, and he felt that the results compared very favourably with those following combined therapy. Previously he used malaria and tryparsamide.

Dr. E. L. HOPKINS said that in 1927 he published a paper on the incidence of syphilis among the admissions to Hanwell Mental Hospital in 1924 and 1925, using the Wassermann test; he was sorry to have to conclude from what he had heard to-day that their results were no good. Among the male admissions the syphilis-rate was 27% in 1924, in 1925, 25%. To follow the matter up, he went through the cases for 1926, and found the incidence-rate had dropped to 22%. The war was thought to have something to do with the high incidence, and as the war date receded the tendency was for the incidence to fall. It would be interesting to see whether that drop would continue. In a large proportion of the cases of general paralysis one could get a history of service in France or other theatre of war.

Dr. FORD ROBERTSON, in further remarks, said he would like to correct a possibly erroneous impression. The Wassermann test, if properly carried out, with due regard to all the difficulties involved and the balancing of the reagents, was a very valuable one from the point of view of specificity, but not from the standpoint of sensitivity. That was why the Wassermann, for mental hospital work, lost its diagnostic value. It did not give positive results in mental patients who nevertheless gave strong positives with the flocculation tests. In the experience of himself and others in Glasgow, the Kahn test was difficult to carry out; some workers had good results with it, others did not. The Meinicke was simpler to carry out, and yielded consistent results, but it suffered from over-sensitivity.

It was necessary that these tests be carried out by someone who was thoroughly conversant with the technical procedure; none of them could be conducted in a haphazard manner. Just as much care was needed for the flocculation tests as for the Wassermann.

The PRESIDENT invited members to say something about private patients. He had been told that the incidence of general paralysis and syphilis cases had fallen more among private than among hospital patients.

Dr. G. W. SMITH said that during the last fifteen years it had been very rare to find general paralytics in private asylums in London. At an earlier period such cases were as common in those institutions as in others.

Dr. RAMBAUT agreed there was now a decrease in the number of cases of syphilis in private mental hospitals.

Dr. RUSSELL said that at his hospital there had been examinations carried out consistently on practically every new admission for the past twelve years by the Meinicke test, and those who might possibly be positive had been controlled by a Wassermann test done at Leeds University. Only on two occasions had the findings been contradictory.

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Dr. PRENTICE, in reply, said he agreed with Dr. Ford Robertson that for examinations of the cerebro-spinal fluid the Wassermann was the better test; he himself depended upon that more than upon the flocculation test.

In reply to Dr. FitzGerald, he thought the position could be taken to be that, done by the proper people, most of the tests were very good, especially in the hands of those who originated them. It was foolish, he thought, to say one test was good, and another was no good. For example, Meinicke used the Kahn himself, also the other tests, in his own laboratory. In capable hands all the tests gave fairly good results.

Dr. Duncan disapproved of nurses being allowed to withdraw blood for tests; the Meinicke reaction was a micro-reaction, and needed only a prick of the finger. At this institution the staff liked the nurses to do all they could in these small ways, and by taking notes, to co-operate with the medical staff. He would not allow nurses to take venous blood or cerebro-spinal fluid; that was a medical duty.

In answer to Dr. Stewart, the statistics he gave referred to the time before and after the institution of the serum test, 1926–7. The 1924 percentages were about the same. They had no private patients.

He was glad to hear of Dr. Pool's successful results. Those who had worked with malaria for many years knew how difficult it was, and if a better method were brought in he would willingly use it.

He rather regretted that the discussion had degenerated into a criticism of things serological; he had hoped to hear more about therapy.

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Part III.—Epitome of Current Literature.*

1. Anatomy and Physiology.

Associated Facial, Vocal and Respiratory Components of Emotional Expression: An Experimental Study. (*Journ. Neur. and Psychopathol., vol. xvii, p.* 241. *Jan.*, 1937.) *Magoun, H. W., et al.*

The writers carried out experiments on monkeys and cats. They found that co-ordinated facial and vocal activity closely resembling that seen and heard during the expression of unpleasant emotion by the normal animal were obtained in response to electrical stimulation of a localized area in the mid-brain and pons of the lightly anæsthetized monkey and cat, and of the acutely decerebrate cat. The anterior portion of the hypothalamus was also found to be reactive in the cat. In both animals the reactive area in the midbrain includes the central grey matter of the aqueduct and a bridge of tissue extending from this region through the dorsal part and into the lateral and ventral portions of the tegmental area. Responses from the decerebrate cat showed that the effects were, partly, independent of afferent projections to the thalamus or cortex, and it would appear that within the reactive area there exists an efferent pathway or system for eliciting co-ordinated facial, vocal and respiratory activity during the expression of emotion in these animals. G. W. T. H. FLEMING.

The Cerebral Circulation: Some New Points in its Anatomy, Physiology and Pathology. (*Journ. Neur. and Psychopath., vol. xvii, p.* 193, *Jan.*, 1937.) *Putnam, T. J.*

The writer summarizes the present position and points out that the cerebral arteries are not end-arteries, but possess a rich system of anastomoses. The liability of the brain toward vascular disturbances is due rather to the sensitiveness of the parenchyma than to deficiency in blood-supply. Different regions in the brain are characterized by marked differences in pattern of blood-supply. This the writer considers may be of importance in the localization of disease processes. The cerebral vessels are under vasomotor control by the sympathetic, but react much more strongly to chemical stimuli than to nervous impulses. Carbon dioxide is a particularly active vaso-dilator. The role of vasospasm in epilepsy and migraine is doubtful. A diffuse loss of myelin may be produced by a mild general anoxæmia and local loss of myelin by a mild local asphyxia, e.g., following venous thrombosis. The lesions which result simulate closely those of " perivenous encephalomyelitis ", multiple sclerosis and diffuse sclerosis. The writer suggests that these diseases are thrombotic in origin. Partial anoxæmia of the cortex leads to a loss of nerve-cells without softening. Evidence is advanced that the parenchymal atrophy in general paralysis is a similar diffuse process, the result of endothelial hypertrophy in the capillaries. G. W. T. H. FLEMING.

* A number of abstracts in this section are reproduced from *Chemical Abstracts* by kind permission of Prof. Crane, of Ohio University, to whom the Editors wish to express their thanks.

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2. Psychology and Psychopathology.

The Psychoneuroses and Neuroses. (*Amer. Journ. Psychiat., vol. cxiii, p.* 107, *July*, 1936.) *Yarkin, J. C.*

The writer studied 100 cases of anxiety neurosis, conversion hysteria, anxiety hysteria, compulsive-obsessive reactions and neurasthenia.

He found a neuropathic family history in 44%, and a psychopathic in 16%. The personality was neurotic in 75% and psychopathic in 2%. In general, the complexity of the causes increased in the following order: anxiety neurosis, conversion hysteria, anxiety hysteria and compulsive-obsessive reactions.

Attempts at compromise formation and partial analysis seemed to be the most effective methods of influencing underlying ætiological factors, especially in anxiety hysteria and in compulsive-obsessive reactions. In the latter group, partial analysis is the only method found to possess worth-while value.

G. W. T. H. FLEMING.

Reality and the Unconscious. (*Psychoanal. Quart., vol. vi, p.* 23, *Jan.*, 1937.) *French, T. M.*

Neuroses and psychoses are not able to ignore pain. Dreams are able to ignore unpleasant reality only in so far as the state of sleep itself in proportion to its depth is able to absorb the pain. If a dream or symptom is unable to provide reassurance by reference to the actual experiences of the patient, distortions of reality in the direction of wish-fulfilment will be followed by a compensatory tendency to correct the distortion in subsequent material.

The fundamental differences that distinguish rational waking behaviour from neuroses and dreams are based upon the quantitative relationship between the synthetic capacity of the ego and the intensity of the conflict. Due to the ego's inadequate synthetic capacity neuroses and dreams are usually able to deal with the conflict only in a fragmentary way, and tend to repeat in a stereotyped manner reactions to previous traumatic experiences. Rational behaviour requires an ego span sufficient not only to view one's situation as a whole, but also to enable one to pay attention to differential criteria so as to be able to learn from past mistakes instead of repeating them. STANLEY M. COLEMAN.

Three Criminal Types as Seen by the Psycho-analyst. (*Psychoanal. Rev., vol. xxiv, p.* 113, *April*, 1937.) *Alexander, F., and Saul, L. J.*

The first case presented concerns a youth of 19, who committed a double murder. In the course of a trivial quarrel he shot his younger brother and also his brother's friend. The psychological problem consists fundamentally in how this weak, introverted, not especially aggressive youth with a constant feeling of inferiority committed a deed of which no one who knew him believed him capable. Analysis revealed a fundamental split in his character—a struggle between masculine demands and the passive infantile need for dependence. His brother, through his physical superiority, had intensified this conflict, and in him he had killed the stronger person whom he envied because of his own masculine ambitions. In the friend he killed the person whose place with his brother he envied because of his own passive feminine feelings. The crime was a desperate effort to resolve the conflict between the two opposing instinct-demands of his personality by an action.

The second case describes a 21-year-old youth, who during the previous five years had a court record of four arrests. The charges were successively—indecent exposure, larceny of a car, disorderly conduct (peeping into windows), and burglary. Analysis revealed him to be what Alexander calls the " tough on the outside, soft on the inside type ", and the trend towards criminality developed out of the need to show his toughness and superiority, which he felt he could not do in normal

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competition because of his infantile receptive dependent demands; the surface show of strength and independence was in reality a flight away from these.

For reasons of discretion the third case presentation is not published.

STANLEY M. COLEMAN.

Scopophilic Instinct and Identification. (*Int. Journ. of Psycho-analysis, vol. xviii, p. 6, Jan., 1937.*) *Fenichel, O.*

For the unconscious to look at an object may mean to devour it, to grow like it (be forced to imitate it) or, conversely, to force it to grow like oneself. The goal of the scopophilic instinct is determined by two tendencies: (*a*) the impulse to injure the object seen, and (*b*) the desire to share by means of empathy in its experience. Scopophilia is a pregenital component of the sexual instinct, and the object relation at this level is one of incorporation, a precursor of love and hate. The underlying tendency may be formulated as follows: "I wish what I see to enter me." This process of ocular introjection is strongly tinged with oral sadism, and is an example of the most primitive form of identification.

The magic glance is a fixed and rigid stare (snake, basilisk, evil eye, hypnotist). Here the eye is a phallic symbol and the fixed gaze stands for the penis in erection. The punishment for libidinal looking is to be turned rigid or into stone (moon-struck, Lot's wife, head of Medusa, etc.). The rigidity of a person turned into stone stands for the fixed gaze and the rigidity of the whole muscular system of a person fascinated by something he sees and signifies erection or (death and) castration. Ultimately the terrible objects of the scopophilic instinct, identification with which takes place by means of a look and upon which are projected one's peculiar bodily sensations, are the parents in the primal scene and above all their genitals.

STANLEY M. COLEMAN.

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3. Psychiatry.

Neurocirculatory Reactions in the Psychoneuroses Studied by the Schneider Method. (*Amer. Journ. Psychiat., vol. cxiii, p. 567, Nov., 1936.*) *McFarland, R. A., and Huddleson, J. H.*

The writers investigated the Schneider index in 503 psychoneurotic patients, 83 schizophrenics, 71 manic-depressives, 90 organic neurologic patients, 134 athletes, and 191 unselected control subjects.

The mean score for the psychoneurotics was 7·9, the manic-depressives 8·2, the schizophrenics 8·7, the organic patients 10·2, the unselected controls 12·6, the athletes 14·8. If a mean score of 9 or below is taken as indicative of cardiovascular unfitness, more than 50% of the psychoneurotic and psychotic patients come within this category, while only 18% of the controls and 5% of the athletes do so. The pulse-rates for the patients were on the average higher than in the control groups. The mean systolic and diastolic blood-pressures of the patients did not differ greatly from those of the controls, except that a large percentage of the patients showed a fall in systolic pressure on standing, while the controls generally showed a small increase with little deviation. Cases of conversion hysteria scored higher than the other psychoneurotics, whilst anxiety states and neurasthenia scored lowest.

G. W. T. H. FLEMING.

Functional Changes in the Patellar Reflex as Seen in the Psychoses. (*Amer. Journ. Psychiat., vol. cxiii, p. 546, Nov., 1936.*) *Strecker, E. A., and Hughes, J.*

The writers, bearing in mind the recent work of Sherrington *et al.* on summation of excitation on the surfaces of anterior horn cells and in internuncial neurones, investigated the effects of mental illness on the excitability of the reflex pathways or synapses of the spinal cord. The knee-jerk and its reinforcement in unselected psychotic patients were studied. A control group of 30 normals was also investigated. Manic-depressive depressed and involutional patients showing symptoms

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of agitation and depression gave large reflex responses. As the mental symptoms subside, the size of the reflex decreases. Agitated and depressed patients are unable to reinforce a maximal patellar reflex response; this is a phenomenon of occlusion. In other words there are so many stimuli coming down the spinal cord from higher cerebral centres that a maximal response takes up all the available neurones. Hence reinforcement of a maximal response fails because of occlusion. These patients can still reinforce a small patellar reflex. Hypomanic patients give unpredictable reflex responses. Schizophrenic patients give responses similar to those of normal individuals. G. W. T. H. FLEMING.

The Syndrome of Episodic Confusions. (*Amer. Journ. Psychiat., vol. cxiii, p.* 625, *Nov.*, 1936.) *Kasanin, J.*

The writer describes two cases of what Kleist has so well described as episodic confusion. He (Kleist) pointed out its possible relationship to epilepsy and migraine and its constitutional nature. He also considered that the disorder was due to a functional disturbance of the nerve elements in the region of the third ventricle.
G. W. T. H. FLEMING.

The Endocrine Glands from the Point of View of Neuropsychiatry [*Les glandes endocrines du point de vue neuro-psychiatrique*]. (*Ann. Méd. Psych., vol. xv,* (*i*), *p.* 394, *March*, 1937.) *De Loverdo, G.*

Endocrine dysfunction, by disturbing the humoral equilibrium, has an action on the whole organism and is nearly always accompanied by some degree of psychical disorder. The author cites examples in cases of thyroid, gonad, pituitary and suprarenal disease. Post-influenzal depression and inertia are due to the involvement of the suprarenal, which is especially sensitive to this infection. The importance of a full knowledge of endocrinology in the treatment of mental disorder is stressed. STANLEY M. COLEMAN.

Periodic Nervous and Mental Syndromes Exclusive of Manic-depressive States [*Syndromes périodique nerveux et mentaux en dehors des états maniaques dépressifs*]. (*Ann. Méd. Psych., vol. xv* (*i*), *p.* 641, *April*, 1937.) *Baruk, M. H., et Gevaudan, Mlle.*

Evidence is brought forward to demonstrate that periodicity may occur in all varieties of psycho- and neuropathic syndromes, and that it cannot be limited to the manic-depressive psychosis. Further, in somatic, neuro-vegetative and cerebro-spinal disorders manifestations of periodicity are frequently to be met with.

It is concluded that periodicity is a fundamental feature of the physiology of the nervous system, and that it can no longer be considered a problem of purely psychological or psychiatric interest. STANLEY M. COLEMAN.

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4. Neurology.

The Ætiology of Headache. (*Bull. Neur. Inst. New York, vol. vi, p.* 12, *Jan.*, 1937.) *Brewer, E. P.*

Ventriculography was performed 86 times on 77 patients. In a large number of instances introduction of air into the ventricles did not cause headache. Headache is often seen to appear after the withdrawal of fluid and to be relieved by the injection of air. Headache occurred as often in patients who did not have as in those who did have intracranial neoplasm. It occurred especially often when the intracranial pressure was high and was very frequently in the frontal region. It is probable that headache is produced by alteration in the intraventricular pressure.
T. E. BURROWS.

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Huntington's Chorea : Its Incidence in the Scottish Highlands. (*Caled. Med. Journ., vol. xvi, p. 31, April, 1937.*) *McWilliam, W.*

The writer found cases of Huntington's chorea confined to one fishing village on the east coast of Ross. All have the surname of either Patience or MacLeman, and appear to be descendants of fishermen who have drifted up north from the district round the Wash. It is interesting that American authorities trace most of 962 choreics back to four families who appear to have come from the east coast of England round the towns of Boston and Stamford. G. W. T. H. FLEMING.

Prolonged Fever Following the Removal of Large Tumours from the Posterior Cranial Fossa. (*Bull. Neur. Inst. New York, vol. vi, p. 33, Jan., 1937.*) *Ehrlich, W.*

The author reports six cases of fever following the removal of a large glioma in the cerebellum, which lay in some cases in the midline, and in others in the hemispheres. In all cases the removal was accomplished by electro-surgical methods, and in all the wound healed without complication. The fever began within one to four days of the operation, reaching 103° or 104° in the evening, and falling to 99° or 101° in the morning. It persisted for more than five weeks in all patients, gradually falling throughout that period to normal. Four of the six patients appeared to be unaffected by the fever, and two complained of headache and other discomforts. No satisfactory explanation for the fever could be found in spite of repeated physical examinations, blood-cell counts, blood-culture and examination of the cerebro-spinal fluid. All the patients recovered. It is suggested that the fever might result from some profound disturbance in the circulation of the cerebro-spinal fluid. T. E. BURROWS.

---

5. Pharmacology and Treatment.

The Roentgen Treatment of Tumours of the Brain in the Operating Room by Direct Radiation through the Open Wound. (*Bull. Neur. Inst., New York, vol. vi, p. 19, Jan., 1937.*) *Elsberg, C. A., Davidoff, L. M., and Dyke, C. G.*

Following experimental roentgen radiation of the brain through the open wound in 16 *Macacus rhesus* monkeys, in order to determine the dosage, the method was applied to 18 patients with intracranial tumour. There was no evidence that there were any harmful effects as a result of the roentgen therapy. T. E. BURROWS.

Pharmacological Action of Three Derivatives of Choline. (*Compt. rend. Soc. Biol., vol. cxxiv, pp. 276–9, 1937.*) *Wispelaere, M. de.*

Acetyl-β-methylcholine (I), β-methylcholine ethyl ether (II), and β-ethylcholine (III) have a hypotensive action, respectively 20, 2 and 10 times that of acetylcholine (IV). The heart-slowing action of I, and to a lesser extent that of II and III, is much more prolonged than that of IV. Atropine suppresses or inverts the hypotensive action of II and III and suppresses the action of small, but not large, doses of I. All 3 are intense excitants of the reflexogenic chemosensitive receptors of the carotid sinus. (Chem. Abstr.).

General Paresis Treated by Mosquito-inoculated Vivax (Tertian) Malaria. (*Amer. Journ. Psychiat., vol. cxiii, p. 619, Nov., 1936.*) *Kusch, E., Milam, D. F., and Stratman-Thomas, W. K.*

The writers studied a group of 72 cases of general paresis treated with mosquito-inoculated vivax malaria, and compared the results with those of a former group of 363 cases treated with blood-inoculated malaria. They found that the course of vivax malaria is milder and better borne when induced through mosquito bite than when induced by blood inoculation.

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Malaria induced by mosquito bite apparently also gives better results than that produced by direct inoculation and is the method of choice.

G. W. T. H. FLEMING.

The Effect of Artificial Fever on the Clinical Manifestations of Syphilis and the Treponema pallidum. (*Amer. Journ. Psychiat., vol. cxiii, p.* 517, *Nov.*, 1936.) *Neymann, C. A.*

The writer draws attention to the fact that temperatures of 105·8° F. and above maintained for at least two hours kill most of the *Treponemata pallida* in the human body, so that all chancres and skin-lesions heal promptly after such treatment. Those treponemata which reach the glands become heat- and chemo-resistant so that arsenicals and heavy metals have to be combined with pyreto-therapy. A patient with a negative or partially-positive blood serology treated by means of electropyrexia immediately yields a strongly positive serology, probably due to the massive destruction of treponemata. The results with electropyrexia in the treatment of general paralysis are every bit as good as those with malaria, if not better, whilst the death-rate is only 2·5%. Burns should not occur. The chlorides lost during treatment are best replaced by giving ·6% sodium chloride to drink.

G. W. T. H. FLEMING.

Delirious Episodes Associated with Artificial Fever. (*Amer. Journ. Psychiat., vol. cxiii, p.* 191, *July*, 1936.) *Ebaugh, F. G., Barnacle, C. H., and Ewalt, J. R.*

The writers used the Kettering hypertherm on 200 patients, including 34 paretics. 108 patients developed 331 delirious episodes amongst the 1,324 fever sessions. Severe deliria were rare. 80% of the deliria occurred during the first session. The general average duration of the delirium was 1¼ hours. The reaction of the patient to the fever depends to a large extent upon the individual equipment and experience of the subject. Emotional instability and personality problems render a patient susceptible to delirious reactions. The writers feel that cerebral œdema is present during the delirious reactions, and that it is particularly evident in the moderate and severe types. The most characteristic and consistent signs of cerebral œdema are manifest in the paretic group.

G. W. T. H. FLEMING.

Clinical Studies in Post-Lumbar Puncture Headaches. (*Amer. Journ. Psychiat., vol. cxiii, p.* 639, *Nov.*, 1936.) *Heldt, T. J., and Whitehead, W. S.*

The writers have evolved a method of preventing the headache which is so troublesome after lumbar puncture. A small piece of sterile catgut is pushed in by the stylet after all the fluid required has been withdrawn and left in the small hole in the dura. The patient is allowed to get up after ten minutes.

There was still a considerable amount of backache, aching of the legs and headache. There was often a rise in temperature of 1° F. for 2–3 hours. These symptoms the authors feel were due to mild meningeal irritation caused possibly by the influence of the foreign body introduced.

G. W. T. H. FLEMING.

The Effect of Pilocarpine on Neurogenic Urinary Retention. (*Bull. Neur. Inst. New York, vol. vi, p.* 154, *Jan.*, 1937.) *Dean, J. S.*

The author reports 19 cases of urinary retention resulting from disease of the nervous system and treated by injections of pilocarpine. Within five days of commencing treatment 17 of the patients voided urine spontaneously. In 15 of these catheterization was rendered unnecessary, but in 4 cases catheterization was continued to prevent distension.

In 7 of the cases abstinence symptoms developed within 24 hours of stopping the treatment, but in 8 of the patients no change occurred in 24 hours, and in 6 cases normal micturition occurred within 2 days of stopping the treatment, and this continued as long as the patients were in hospital.

T. E. BURROWS.

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The Use of Benzedrine Sulphate in Post-Encephalitic Parkinson's Disease. (*Journ. Amer. Med. Assoc., vol. cviii, p.* 1765, *May* 22, 1937.) *Solomon, P., Mitchell, R. S., and Prinzmetal, M.*

Twenty-eight patients with post-encephalitic Parkinson's disease, 10 patients with arterio-sclerotic Parkinsonism and 22 patients with psychoneurosis exhibiting asthenia were given benzedrine sulphate for periods varying from 4 weeks to 16 months. The dose varied from 10 mgrm. to 160 mgrm. orally per day, either alone or in combination with scopolamine or stramonium. The study was controlled by the substitution of inert pills in 19 of the patients, and the effect of ephedrine was compared with that of benzedrine in 12 cases.

The results in the post-encephalitic group which signify benefit, derived from benzedrine alone or in combination with scopolamine or stramonium, were as follows: Subjectively increase in energy in 25 out of 26 who complained of lack of energy. Disappearance of drowsiness in 23 out of 24. Increased feeling of strength in 19 out of 26. Decreased or abolished tremor in 7 out of 15. Subjectively decreased muscular rigidity in 20 out of 26. Disappearance of oculogyric crises in 6 out of 8, and greatly diminished frequency and severity in the other 2. Increased ability to work in 22 out of 28. The effect of benzedrine alone was not equal to that obtained with stramonium or scopolamine. Benefit obtained with ephedrine was equal to that of benzedrine in only 3 out of 12 cases. None of the arterio-sclerotic patients was benefited by benzedrine, and there was improvement in only 2 out of 22 among the psychoneurotics. The following untoward symptoms are mentioned as having occurred, especially in the arterio-sclerotic and psychoneurotic groups: insomnia, dizziness, light-headedness, excitement, agitation, nausea, anorexia, vomiting, dryness of the tongue and hallucinations. T. E. BURROWS.

The Stimulating Action of Benzedrine Sulphate. (*Journ. Amer. Med. Assoc., vol. cviii, p.* 1770, *May* 22, 1937.) *Davidoff, E., and Reifenstein, E. C., jun.*

Benzedrine sulphate in doses of 10 to 30 ngrm. daily, was given to a number of individuals representing each of the following five groups: normal, self-absorbed organic, self-absorbed dementia præcox, depressed manic-depressive and depressed psychoneurotic. None of the subjects except two of the normal group were acquainted with the nature of the medication. Of 10 normal individuals, 7 showed elevation of mood, 7 over-talkativeness, 10 increased motor activity, and 10 increased general efficiency. Five exhibited increased irritability and 3 complained of fatigue. Two suffered from lassitude after the medication was discontinued, and 2 others requested continuation of the drug.

A great variety of other symptoms, mostly unpleasant, are reported, including fatigue, malaise, anxiety, dizziness, aggressiveness and confusion. Of 10 patients in the organic group, 5 exhibited elevation of mood, 7 over-talkativeness, 8 increased motor activity, 8 increased general efficiency, and 3 irritability.

The most consistent improvement was noted in alcoholic and traumatic cases. In the group of 25 cases of dementia præcox, 4 manifested elevation of mood, 7 over-talkativeness, 11 increased motor activity, and 9 increased general efficiency. Five showed increased irritation. Three diagnosed as catatonic were improved and paroled. There was no change in the structure of the psychosis in any of the cases.

Of the depressed manic-depressives 2 showed elevation of mood, 6 over-talkativeness, 6 increased motor activity, 6 increased general efficiency, and 6 increased irritability. One patient became more depressed and attempted suicide. In the group of 10 psychoneurotics, 3 showed elevation of mood, 4 increased motor activity, 2 improved general efficiency and 6 became talkative. The psychoneurosis in each case remained unchanged. T. E. BURROWS.

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6. Pathology and Biochemistry.

The Cholesterol Content of the Cerebro-spinal Fluid. (*Arch. Psychiat., vol. cii, pp.* 147–96, 1934; *Ber. ges. Physiol. exptl. Pharmakol., vol. lxxxiii, p.* 380.) *Holthaus, B., and Wichmann, B.*

The cholesterol content was determined by a modification of the Bloor, Allen and Pelkan method for blood-serum, with a Pulfrich photometer. The Hauptmann saponin-hæmolysis reaction, the sugar content, the Kafka albumin relation, the NaCl content and the gold sol reaction were determined on the cerebro-spinal fluid of 383 patients. Cholesterol was always present, normal values in 56 patients ranging between 0·3 to 0·6 mgrm. %. With progressive paralysis, 39 patients showed 0·36 to 0·92 mgrm. %; 13 patients with fever showed 0·4–1·03 mgrm. %. No changes were observed in 4 cases of tabes dorsalis, and no significant changes in 6 patients with meningitis. Slight increases were observed in 28 of 56 epileptics. Decreased cholesterol was observed also in hydrocephalus. Increases were observed also in arterio-sclerosis, encephalitis, schizophrenia, manic-depression, myelitis and migraine. An increase in cholesterol was observed when definite increase in lipoids occurred in the nervous system or when there were changes in the blood-cerebro-spinal fluid volume. JAMES C. MUNCH (Chem. Abstr.).

Newer Researches Regarding the Bromine Picture in the Blood of Patients with Psychosis. (*Klin. Wochenschr., vol. xv, pp.* 1832–3, 1936.) *Meier, C. A., and Schlientz, W.*

Br. determinations were made by the method of Liepert-Watzlawek. The blood is ashed with chromic acid in a closed system and the Br. titrated as bromate. The bloods of a large series of psychotic patients were found to contain 75–233 mgrm. with an average of 130 mgrm. % Br. No relation between the type of psychosis and Br. value was found. The higher values reported in the literature probably represent a previous Br. therapy. The table salt in Zurich contains half as much Br. as that in Vienna. This may explain the lower Br. values in patients who live in Zurich. W. MENDELSOHN (Chem. Abstr.).

The Clinical Application of the Phosphatide-splitting Property of Serum from Normal and Psychotic Individuals. (*Klin. Wochenschr., vol. xvi, pp.* 59–60, 1937.) *Freund, E., Lustig, B., and Heimann, M.*

Mixtures of serum and brain emulsion were incubated for 24 hours at 37°. With normal serum the inorganic P increased from 0·1 to 0·95 mgrm. % (av. 0·38). With serum from patients with general paresis, epilepsy, paranoia and schizophrenia, in 60–66% of the cases, the increase in inorganic P was greater than with normal serum. The average increase was 1·71 mgrm. %.

W. MENDELSOHN (Chem. Abstr.).

The Determination of Vitamin C Content in the Human Brain by Means of the Dental Test. (*Klin. Wochenschr., vol. xvi, pp.* 93–6, 1937.) *Kalnins, Viktors.*

Scurvy was induced in guinea-pigs by feeding a vitamin C-free diet. After administration of various amounts of ascorbic acid for 16 days, histologic examination of the teeth was made. In this way standards of healing were obtained. The effect of feeding brain emulsion as compared with ascorbic acid serves as the basis for this test. In 1 grm. adult and infant brain there was 1·15 and 0·5 mgrm. ascorbic acid respectively. W. MENDELSOHN (Chem. Abstr.).

The Phospholipides of the Brain, Kidneys and Heart of White Rats in Experimental Hyperthyroidism. (*Endocrinology, vol. xxi, pp.* 101–8, 1937.) *Weil, Arthur.*

When thyroid powder is fed to rats there is a decrease in body-weight and an increase in the weight of the kidneys and heart. There is a decrease in water, an increase of AcMe-sol. extracts in brain, kidneys and heart, an increase in alcoholic

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extracts in kidneys and heart and a decrease in the brain. The total P and the P of the different extracts of the three organs are increased proportionately to the increase in phospholipides and weight of the organ. There is no qualitative change in the phospholipides. FELIX SAUNDERS (Chem. Abstr.).

Further Investigations on the Blood-brain Barrier. The Significance of the Electrical Charge and the S-potential in the Problem of the Blood-brain Barrier and Capillary Permeability in General. (*Journ. Immunol., vol. xxxii, pp.* 97–117. 1937.) *Friedemann, Ulrich.*

After intravenous injection of 1–7 mgrm. into guinea-pigs weighing 300 grm., all basic aniline dyes with the exception of safranine stain the brain. Some acid dyes (acid fuchsin, orange, bordeaux, eosin, uranin) do not stain the brain in the amounts used. Some others (trypan blue, Congo red, water blue, alizarin blue-S, indigo di- and tetrasulfonate) stain in amounts of 20 to 80 mgrm. The difference between the acid and basic dyes is partly due to a difference in their affinity for brain tissue, which was tested by determining the minimum staining dose *in vitro*. The cerebral capillaries are more permeable to basic than to acid dyes. This is determined by the ratio between the minimum staining concentrations *in vivo* and *in vitro*. The permeability of the cerebral capillaries to dyes is independent of their chemical constitution, size and lipoid solubility, but depends on their electrical charge. Staphylococcus toxin acts rapidly without any incubation period. It is unable to pass the blood-brain barrier. It carries a negative charge at the pH of the blood and the iso-electric point is near 6·2. A method was devised to compare the *S*-potentials of toxins, with which the capacity of toxins to pass the capillary system is correlated. A theory is advanced that the selective permeability of the capillary system in general and the blood-brain barrier in particular is due to an irreciprocal permeability. A. R. BEEBE (Chem. Abstr.).

An Investigation of the Absorption of Ultra-violet Light by Cerebro-spinal Fluid in Various Disease States. (*Journ. Neur. and Psychopathol., vol. xvii, p.* 213, *Jan.*, 1937.) *Skinner, E. F.*

The writer continued his work on absorption spectra in the spinal fluid. In his first series of 50 cases he found that cases of tuberculous meningitis gave rise to a characteristic change in the curve, as also did general paralysis, but not to such a marked degree. During the course of a disease the curve tended to become steeper if progress was towards fatality. The curve of general paralysis shows a shift towards the region of the long waves. In cases of long standing the hump on the curve disappears and the curve becomes merely a steeply-sloping line, closely resembling the curve found in tuberculous meningitis. After malarial treatment the curve approximates to the normal. The shape of the curve does not depend on a pleocytosis. Removal of all the salts by dialysis eliminated the hump from the curve and diminished the amount of absorption. Selective absorption gives way to a general absorption beginning about 2350. Normal fluid absorbs the ultra-violet waves in a region between 2600 and 3150, so causing the " hump " on the curve. Removal of proteins obliterates the hump by increasing the absorption and moves the curve to the right. There appear grounds for thinking that the hump in the normal curve may be due to uric acid, and that the shift to the right in inflammatory conditions is possibly due to an increase in the constituents. The writer suggests that coma may be due to the action of uric acid on neurones.
G. W. T. H. FLEMING.

Fatty Acids of Phrenosin and Kerasin. (*Biochem. Journ., vol. xxx, p.* 100, *Jan.*, 1936.) *Chibnall, A. C., Piper, S. H., and Williams, E. F.*

These authors found that brain contains at least three phrenosins whose acid components are –OH-n-docosanic, -OH-n-tetracosanic, and -OH-n-hexacosanic acids. Phrenosinic (cerebronic) acid is a mixture of these acids. Brain also

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contains at least three kerasins, whose acidic components are n-docosanic, n-tetrasocanic and n-hexacosanic acids.

Evidence that lignoceric acid is a mixture is summarized and the suggestion made that the name should be abandoned. G. W. T. H. FLEMING.

α-Glycerophosphoric Acid and Brain Metabolism. (*Biochem. Journ., vol. xxx, p.* 33, *Jan.*, 1936.) *Johnson, R. E.*

The writer investigated the metabolism of -glycerophosphoric acid in the brain of normal pigeons. He found that the rate of aërobic removal of sodium-glycerophosphate is unaffected by added sodium lactate, but is increased by added sodium pyrophosphate. Added sodium pyruvate has no effect on the aërobic or anaërobic removal of α-glycerophosphate with or without added pyrophosphate. So that the Embdin-Meyerhof scheme does not hold for the pigeon's brain. There is aërobic production of reducing substances from α-glycerophosphate. From solutions in which brain-tissue has respired with α-glycerophosphate the 2–4-dinitrophenylbishydrazone of methylglyoxal and the 2–4-dinitrophenylhydrazone of a compound of probably six carbon atoms is formed. The possibility is discussed that the brain may synthesize oxidatively a compound of six carbon atoms from the α-glycerophosphate. G. W. T. H. FLEMING.

Choline Ester Formation in, and Choline Esterase Activities of Tissues in vitro. (*Biochem. Journ., vol. xxx, p.* 1668, *Sept.*, 1936.) *Quastel, J. H., Tennenbaum, M., and Wheatley, A. H. M.*

The writers found that brain slices respiring in a medium containing eserine produced a substance which caused a powerful contraction of an eserinized leech muscle preparation. This substance was only formed in the presence of eserine, was unstable in dilute alkaline solution, and its action on the leech muscle was counteracted like that of acetylcholine by the presence of morphine. It is considered to be a choline ester, and its rate of formation is greatest in the first hour. Oxygen is necessary for maintenance of this rate at a high level. The presence of glucose greatly increases the rate in phosphate or bicarbonate media when potassium and calcium ions are absent. The addition of potassium and calcium ions decreases the rate in a bicarbonate glucose medium. It is suggested that there is a link between glucose metabolism and choline ester metabolism in tissue slices. The presence of sodium lactate, pyruvate, α-glycerophosphate or glutamate increases the rate of respiration, but their effects are not as great as those of glucose. The presence of sodium succinate does not increase the rate. The addition of sodium fluoride, of choline or of a large excess of acetate ions does not diminish the rate.

G. W. T. H. FLEMING.

Sodium Chloride Content of Cerebro-spinal Fluid in Tuberculous Meningitis. (*Brit. Med. Journ., vol. ii, p.* 111, 1937.) *Ingham, J.*

The writer used a modification of the Whitehorn method of estimating the chlorides. Any possible contamination by chlorides is thus eliminated. He found an average of 715·6 mgrm. in 6 normals, of 633 mgrm. in 10 cases of non-tuberculous, non-meningitic lesions of the nervous system, of 633 in 26 cases of non-tuberculous meningitis. In 84 cases of tuberculous meningitis the average was 510 mgrm., the highest was 550 mgrm. and the lowest 420. G. W. T. H. FLEMING.

Respiration of Brain. (*Biochem. Journ., vol. xxx, p.* 1577, *Sept.*, 1936.) *Dixon, T. F., and Meyer, A.*

The writers investigated the respiration of the striate body, globus pallidus, cerebellar cortex, the cornu ammonis, hypothalamus and medial parts of the thalamus in the presence of different substrates. In the minced tissue, certain areas showed little or no extra oxygen uptake in the presence of glucose or lactate,

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whereas when slices of these regions were used, constant and much higher respiration rates were observed. Cerebellar cortex has greater metabolic activity than any other part of the brain. G. W. T. H. FLEMING.

Studies on Brain Metabolism. I. The Metabolism of Glutamic Acid in Brain. (*Biochem. Journ., vol. xxx, p.* 665, *April*, 1936.) *Weil-Matherbe, H.*

The only amino-acid oxidized by brain is *l*(+)glutamic acid, which is oxidized to *α*-ketoglutaric acid and ammonia, and further to water and carbon dioxide. The enzyme responsible for the oxidation of *l*(+)glutamic acid does not attack *d*(−) glutamic acid so long as it is bound in the cell or to some constituent of the cell, probably a lipoid. In solution, however, the specificity is changed and *d*(−) glutamic acid alone is oxidized. The ammonia derived from the de-amination of *l*(+)glutamic acid disappears in secondary reactions leading to and beyond glutamine. The reaction *l*(+)glutamic acid → *α*-ketoglutaric acid is reversible. The existence of an ammonia-binding mechanism is shown leading from glucose via pyruvic acid and *α*-ketoglutaric acid to *l*(+)glutamic acid, glutamine and further to an end-product. *In vivo* the glutamic acid de-aminase is rather concerned with the synthesis than with the breakdown of glutamic acid.

G. W. T. H. FLEMING.

A Study of Sugar-Tolerance Tests in Two Hundred Patients with Convulsions. (*Bull. Neur. Inst. New York, vol. vi, p.* 62, *Jan.*, 1937.) *Drewry, H. H.*

In 200 cases with convulsions, dextrose tolerance tests were done before a diagnosis had been reached and the results compared when the series was completed. 93 patients had sugar curves with a peak below 150 mgrm. per 100 c.c., and 27 with a peak below 120 mgrm.; 3 patients had blood-sugar levels below 50 mgrm., and all 3 were found to have an adenoma of the pancreas. 24 had levels at one time or another between 50 and 70 mgrm., and all without definite symptoms of hypoglycæmia at the time the blood was taken.

Encephalography caused a rise in the sugar curve, which tended to return to the initial level as time passed.

It is concluded that factors influencing the blood-sugar level are so numerous and so complex that interpretations of the blood-sugar levels or sugar-tolerance curves must be made with caution. T. E. BURROWS.

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JOURNAL OF MENTAL SCIENCE

CONTENTS FOR JULY, 1937

PAGE

Original Articles—

Mental Observation Wards: A Discussion of their Work and its Objects; by *E. U. H. Pentreath, M.R.C.S., L.R.C.P., D.P.M., and E. Cunningham Dax, B.Sc.Lond., L.M.S.S.A., D.P.M.* 347

"Temporary" Treatment: An Analysis of Thirty Cases; by *L. H. Wootton*, M.C., *M.B., D.P.M., and Louis Minski, M.D., M.R.C.P.* 366

Acetarsol in the Treatment of Late Congenital Syphilis Amongst Mental Defectives; by *K. C. L. Paddle*, M.C., *M.R.C.S., L.R.C.P., D.P.M.* . 372

Acrocyanosis; by *Edward Samuel Stern, M.A., M.D., B.Ch.Cantab., M.R.C.P., D.P.M.* 408

A Note on Some Vasomotor Disturbances in Schizophrenia; by *Louis Minski, M.D., M.R.C.P.* 437

Schema for the Examination of Organic Cases; by *W. Mayer-Gross, M.D., and E. Guttmann, M.D.* 440

The Iron, Copper and Manganese Content of the Human Brain; by *A. H. Tingey, M.A.* 452

An Undecided Compensation Claim Arising from the Suicide of a Voluntary Patient; by *K. K. Drury*, M.C., *B.A., M.D., D.P.M., and C. E. J. Freer* 461

Syphilis in Mental Hospital Practice; by *David Prentice, M.B., Ch.B., D.P.M.* 472

Epitome of Current Literature 478

1. Anatomy and Physiology.—2. Psychology and Psychopathology.—3. Psychiatry.—4. Neurology.—5. Pharmacology and Treatment.—6. Pathology and Biochemistry.

CONTENTS OF SUPPLEMENT

The Royal Medico-Psychological Association: Quarterly Meeting.—Notice by the Secretary of the Research and Clinical Committee.—Notices by the Honorary Librarian.—Notices by the Registrar.—Notices by the General Secretary.—Notices of Meetings.

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Part I.—Original Articles.

SOME OBSERVATIONS ON THE CARE OF THE INSANE.

THE PRESIDENTIAL ADDRESS DELIVERED AT THE NINETY-SIXTH ANNUAL MEETING OF THE ROYAL MEDICO-PSYCHOLOGICAL ASSOCIATION, HELD AT AYR, JULY 7, 1937.

By DOUGLAS MCRAE, M.D., F.R.C.P.E.,
Medical Superintendent, Glengall Hospital, Ayr.

MY address to you to-day consists of observations on a number of subjects connected with the care of the insane, which may be of interest to you either because they are specially associated with Scottish administration, or because of their topicality at the present time.

BOARDING OUT.

The boarding out of some of the insane in private dwellings has been for very many years an organized part of the Lunacy Administration of Scotland. It was an outcome of the recognition of the fact that all persons who are insane do not require care and treatment in asylums, either in their own interests or in those of the public. The type suitable for this method of treatment and care were "broadly described as incurable, harmless and easily managed". By providing for some of the insane in this way a benefit was believed to be conferred not only on them, but also on those of the insane who require to be treated and cared for in asylums. It was believed also to prove a benefit to the ratepayers, by preventing the needless multiplication or enlargement of asylums.

In 1875, according to the above-quoted writings of Dr. John Fraser, Commissioner in Lunacy for Scotland, the number of pauper lunatics in private dwellings was over 20% of the total number of pauper lunatics on the registers of the Board, and, in the year 1900, this figure had reached the extraordinary proportion of nearly 24% of the total registered number of

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lunatics in Scotland. As Fraser said, it is of importance to know that since 1875, when the exodus of incurable and harmless patients from asylums began to increase, there had been practically no difficulty in finding *suitable* guardians.

Unfortunately it may be said that since the Great War, on account of many economic and social changes that have taken place, there has been a very decided diminution in the number of persons to whom this description might be applicable. It would appear, however, that in more recent years this set-back is being progressively overtaken, and there is little question but that most of the credit for the improvement is due to the enthusiastic and increasing endeavours of the Deputy Commissioners, whose province it is to inspect the boarded-out cases throughout Scotland.

In all fairness to medical superintendents of our mental hospitals, however, I would draw attention to a statement made by one of the Deputy Commissioners in the Annual Report of the General Board of Control for the year 1934.

In seeking to explain the causes of the decrease in the number of patients being boarded out nowadays, he remarks : " It is possible, but it seems unlikely, that the type of patient in our hospitals has changed so much that there are now noticeably fewer patients suitable for boarding out. It seems more probable that, along with others, two important factors have been at work. The first is the economic working of the mental hospital. There is no question but that a considerable amount of work in mental hospitals is done, under supervision, by mental patients. This is a sensible policy, both for therapy and economy, but there comes a time with a proportion of the patients when they could be working in the outer world under guardianship. If this proportion of patients were boarded out, it probably would entail a slightly larger staff in the hospital, with a higher rate of cost for each occupied bed, but it might do something to arrest the rate of increase of building which is so constant a threat at the present time. It appears to me that it would be the wise and far-sighted policy of Hospital Committees to support medical superintendents in giving patients a trial under boarding-out conditions, even although this meant a slight increase of staff and *per capita* rate, for it would not cast on the community the weight over years of the dreary burden of large expenditure on buildings."

In reply to these observations, I may say that at a meeting of the Scottish Division of this Association, held at Inverness twelve years ago, a similar assertion was refuted by several members present, when a Deputy Commissioner read a paper in which it was suggested that medical staffs in asylums were apt to neglect the chronic types of patients ; and that there must be a number of cases suitable for boarding out that were overlooked. It was explained to him even at that time that the type of case being admitted to our asylums was more suitable for "boarding up" than for boarding out ! During the previous twenty-five years I myself had had personal experience of the system in two

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large institutions, and had noted cases returning to them from boarding out, in a state of obvious neglect, several even verminous ; and that commonly when a boarded-out case had become decrepit and unfit for further work, or had developed some physical disease, such as cancer or phthisis, not omitting old age, he was sent back to the asylum. Similarly mental defectives, in those days described as lunatics, were also admitted to swell the death-rate or cause accumulation, should the plea be one of ill-health or of having faulty or other troublesome habits with which the guardian was unable to cope. No mention was ever made of the patient being unfit for further work !

In his paper the author claimed that the future of the boarding-out system in Scotland lay to a large extent in the hands of asylum superintendents ; to which I replied that the asylum physicians could not possibly be held responsible for the type of case that was being admitted to our asylums. Taking into account the fact that a steadily increasing proportion of elderly and decrepit patients and much larger numbers of demented and deteriorated cases are being admitted since that date, it should be obvious that the types in residence have seriously worsened as regards suitability for boarding out or even of usefulness in the institution.

By the end of 1936, excluding mental defectives, of the women resident in Glengall Hospital, 83% were over 40 years of age, 65% were over 50, 40% were over 60 years, and 17% were over the allotted span of life.

It is a very striking fact that some twenty years ago there were only twelve able-bodied male patients who were incapable of useful employment on account of their abnormal mental state. To-day there are 100 male patients who are either too grossly demented to work, or who, on account of their delusional state of mind, absolutely refuse to take part in useful occupation of any sort.

Nearly half the total number of 670 patients resident to-day are over 50 years of age, and in the other half are included 160 mental defectives under that age, for whom, as yet, no other institutional accommodation is available.

I submit, therefore, that the type of patient has changed, and that to a very remarkable degree, and there is no evidence whatever on which to base the assumption that patients are kept in asylums, in preference to being boarded out, because of their usefulness to the institution. A relevant and important fact which must also be borne in mind is that working patients to-day consist almost entirely of the few cases that are convalescing preparatory to being discharged, and of those patients who are the subject of recurrent mental attacks which render them quite unfit for any sort of work, often for many months at a time.

Voluntary Patients.

A voluntary patient should be a person who, in the opinion of the medical superintendent agreeing to admit, is aware of his unsettled mental state and is clearly capable of exercising a reasoned judgment—being free from delusions—

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whose emotional state is not likely to give rise to unreasonable conduct to his own hurt or the danger of others; there being no history or evidence during examination of impulsive acts of violence nor depression amounting to a desire not to live, or prompting the view that those depending on him would be better dead.

The patient must realize that he is placing himself under care and treatment, the nature of which is entirely left to the medical man. It is not a question of volition, for many dangerous acts are performed by the insane while exercising volition of unwarrantable character on account of delusional ideas, emotional instability and inability to exercise a reasonable self-control.

There must be no element of compulsion or coercion. Does the patient wish to come in? Is this wish being freely expressed? I have had cases brought to me that were obviously being coerced, and many that were so facile as to do whatever was suggested, without demur.

Many patients are sent by doctors to be admitted on a voluntary basis because they wish to avoid both the responsibility of certifying and the anxiety of further attendance on the case. They are afraid to continue the case at home, yet shirk the removal of the patient on certificate, and so send the individual to the asylum in the hope that the superintendent may be induced to shoulder the burden of care and responsibility. It may be that on account of the protracted nature of the illness the patient's funds have become seriously depleted and the doctor's bill no longer able to be met, or that the patience or sympathy of the relatives has become exhausted and they too desire to be relieved of their burden.

I am constantly told by relatives that it was the doctor who for long dissuaded them from sending the patient to the asylum, and just as emphatically am I assured by the doctor that it was the relatives who, for an equally long time, refused to take his advice and agree to the patient's removal to a mental hospital, and only in the light of further experience of the case am I in a position to form an opinion as to the value of these assertions.

I am often approached by relatives who wish to bring a patient with a view to admission on a voluntary basis without the knowledge of their own medical practitioner. I naturally refer them to the doctor to ascertain his wishes in the matter. If the case, however, turns up at the asylum in the care of relatives and appears to me to be obviously requiring treatment, I telephone the doctor telling him that one of his patients has come to see me and I get his report of the case. I am then in a position to examine and to decide if the case is to be admitted as a voluntary patient, whether I get the doctor along to certify or the relatives be induced to return home with the patient for a further course of treatment by his own doctor.

It has to be remembered that a voluntary patient is primarily acting on suggestion, and that this can only too readily be transformed into active coercion on the part of friends, who may wish to get rid of the patient, but only

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on a voluntary basis, so as to enable them to secure moneys on the patient's signature to " dividend warrants and similar documents ". This procedure is often urgently stressed by the relatives, quite regardless of the patient's inability to understand the nature of any financial or other transaction in connection with his estate. Similar objection to certification on the part of relatives—especially when the patient has been previously resident in other institutions—transpires when the case has ultimately been certified and the question, involving extra expense, of the appointment of a curator bonis has to be considered.

I have received patients who have been in and out of other asylums as voluntary boarders who exhibit a degree of mental enfeeblement precluding them from taking any intelligent interest in their financial affairs, and obviously certifiable, but who, it would appear, have for years been appending their signature to dividend warrants and similar papers involving disposal of funds, on the instruction of relatives, regardless of the fact, often admitted by the patient, that he does not know why he signed such. I fear therefore that it often happens that the question of finance has more significance in the policy of the relatives than any concern whatever as to the patient's mental welfare. As is often the case in other matters connected with mental illness, the almighty dollar is the ruling factor.

There is a flagrant practice nowadays that, in rate-aided cases where N.H. insurance benefit has been accumulating for months or even years, the relatives press to have the patient discharged to their care to enable them to have payment of the sum accrued—as much as £40 to £50 in some cases—and when this is spent the patient is recertified and returned to the asylum till a further accumulation makes a repetition of the procedure worth while.

The Board say in their circular that " they are clearly of opinion that no person of whom it cannot be said he fully understands and appreciates the voluntary nature of his residence ought to be received in an asylum ". It is difficult to understand how they are able to determine the patient's degree of intelligence when, without having seen him, they issue by post a sanction for his admission, although " recognizing that it must necessarily be left to the discretion of the Medical Superintendent whether the case is certifiable or not ".

In my opinion the patient's application to the medical superintendent and to the General Board for sanction to be admitted should only be made out and signed after the superintendent has seen, examined and agreed to take the patient on a voluntary basis. The procedure is clearly indicated in Section 59 of the Mental Deficiency and Lunacy (Scotland) Act, 1913, which was expressly framed to legalize what I believe had been the practice in some asylums of having a person resident as a " guest " pending the receipt of the assent of the Board to admit the patient as a voluntary boarder. The section runs thus: " If a person desires to submit himself to treatment in an asylum as a

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voluntary boarder under the provisions of Section 15 of the Act of 1866, it shall be lawful for the superintendent to receive such a person on his written application, provided that such person shall forthwith make application to the Board or one of the Commissioners, and that such person shall not be detained for more than three days from the date of reception without the assent in writing of one of the Commissioners". I suggest that it is putting the cart before the horse to have the sanction for the patient's admission forwarded before he has been examined by the medical superintendent and signed his application. It entails the possibility of the patient refusing to enter the hospital or being found unsuitable for admission on a voluntary basis, incurring thereby disappointment, needless expense and much inconvenience to all concerned, especially the relatives, who have been led to expect that there would be no difficulty in having the patient admitted. I have known relatives to hand me an application for the admission of a voluntary patient who was obviously quite unaware of the nature of the document he had been induced to sign.

Many years ago, during his visit, I was asked by a Commissioner it I always made sure that the patient realized that he could leave on giving the usual three days' notice. I assured him that I did, but that I went a step further; after showing the patient his accommodation and getting him clearly to understand that in agreeing to come under my medical care he was faithfully to abide by medical orders and so help me in securing his recovery, it was then explained to him that should he wish to leave at any time I required three days' notice to enable me to send for his relatives, and if necessary two doctors, for the express purpose of his being detained should we all agree that he was not well enough to go home. The patient, thus realizing the position from the outset, never runs the risk of bearing resentment. To those persons who doubt the soundness of this argument I suggest further that there is the legal authority, in the interest of the patient's safety, to detain him against his will, however much he may resent it, for the period of three days.

In referring, in a circular letter, to the subsequent certification of a voluntary boarder the Board says: "Persons who have entered asylums as voluntary boarders are not infrequently found, either on reception or after undergoing mental deterioration during a more or less prolonged voluntary residence, to be in a condition requiring certification. It has come to the notice of the Commissioners that such persons, having entered the asylum as voluntary boarders, and having been subsequently certified and compulsorily detained, often regard themselves, not unnaturally, as having been entrapped by the procedure, and feel a sense of resentment at their voluntary action having been, as it seems to them, taken advantage of to secure their confinement." To meet the situation the Board tender advice thus: "In the case therefore of all voluntary boarders requiring to be certified, and especially in the case of those whose mental state has *not* undergone a marked change since

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reception, the Board strongly recommend that the friends should be advised to remove the patient, on certification, from the asylum in which he has been a voluntary boarder to another asylum." In this paragraph it is difficult to conceive how a case whose mental state has not undergone a marked change since reception should require to be certified. That some might resent being detained is no reason why *all* must be removed to another asylum, since most of the cases may be quite oblivious to the fact either that they are being detained or have been certified. In most cases in district asylums removal to another asylum by the relatives or the local authority is obviously out of the question.

A Few Points on Certification.

The revolt against certification, or, as it is now termed, the "stigma of certification", so actively kept alive by varied propaganda from many quarters —Parliament, platform, press, practising physicians, not excluding neurologists and even lay members of Boards of Control—is not always in the best interests of the patient's welfare or security of his estate. In my opinion it is merely a substitute for the so-called "stigma of insanity" that has for so long served as a label to describe the popular attitude to the whole question of mental disease, and only serves to foment public prejudice and ignorance.

It is extremely uncommon, in my experience, for any patient to exhibit the slightest concern as to whether he is certified or not. In the rare event of a patient voicing any resentment in the matter, it can readily be traced to the disclosures of a disgruntled relative, who complains of being financially handicapped through the invalidity of the patient's signature or some pension disqualification. Again it is the relative who dislikes certification, not the patient.

Another source of objection to certification in recent years is the disclosure to immigration authorities that there is insanity in the family. I have had letters from descendants of former patients imploring me to provide them with a statement to the effect that although the patient had been under my care, he or she was not really a case of insanity; this form of certificate being required by the immigration officials of the country to which the family proposes to emigrate. The day may come when to admit having attended a psychiatric or "nerve" clinic will occupy the same place on the questionnaire of the immigration authorities or assurance companies as insanity or detention in a mental hospital does to-day.

The arch-objector to certification, however, is the criminal case who finds himself undergoing an "indeterminate sentence" in an asylum. He often indulges in recrimination, and avers with unfeigned disgust that he would sooner have stood his trial and undergone a definite term of imprisonment to regain his liberty in due course—a not unreasonable attitude in a man otherwise manifestly insane.

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It is often advanced by the opponents of certification—and their name is legion—that the patient's chances of re-employment on discharge are seriously jeopardized. In all my forty years' experience I have yet to find the case that has not been reinstated immediately he recovered and returned home. It has all along been my practice to place convalescent patients on parole in an open villa for a period of one month, thus giving the relatives time to acquaint the employer of the patient's recovery and fitness to resume his former occupation. Indeed, in the majority of cases the relatives are only too prone to press for the patient's discharge, as the employer is anxiously inquiring how soon he can get the man's services, his post in many cases being kept open for an indefinite period. The recovered case is discharged on Saturday to resume work on Monday morning—the exception in the old days being the alcoholic, who was kept till Monday!

In many cases where the relatives are unable to make a home for the patient, a suitable place in domestic or other service has been secured by the efforts of the members of the hospital staff among their acquaintances. For some years it was quite common for a prospective mistress to ring up the asylum to inquire about the suitability of an applicant who had freely divulged the fact that she had recently been a patient in the hospital. The lady would want to know if the ex-patient could be trusted to take care of children, or otherwise be safe to be left alone in charge of the house. On an assurance being given that the patient was completely recovered, competent and trustworthy, she was promptly engaged. Better the devil you know than the devil you don't! Not a few householders in the county are indebted to this hospital for providing them with conscientious and capable servants.

Many of our former patients visit us periodically, go into the wards and bestow gifts on their friends among the patients and staff. Others often invite a few patients with a nurse to have tea in their homes. From all this it may be seen that such friendly and helpful relations between patients and staff serve to take the place of those voluntary bodies which undertake the "after-care" of the discharged patient.

That asylum detention has no detrimental effect as regards future employment can scarcely be better shown than in the case of a shell-shocked clerk in the office of a public authority. This man, while drinking heavily, was foolish enough to embezzle, and on disclosure made a serious attempt at suicide by poison. Admitted in a state of collapse, he was for a time extremely melancholic, but in the end made an excellent recovery, and as the result of my certificate ascribing his drinking habits to his nervous state rather than as the cause of his mental breakdown and serious default, he received an important post in the Civil Service.

I have had a similar case of a gentleman in the Diplomatic Service who had a sharp attack of mania as the result of a head injury, and after discharge from the mental hospital resumed his responsible occupation on a certificate

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that his recovery was complete and that there need be no recurrence of the symptoms.

Just what constitutes a certifiable case seems to baffle even the General Board, who in their annual report, in referring to the death of a patient by hanging, describe him as a voluntary patient *and* suicidal.

When a neurologist, as it appeared in the report of a medical debate on the so-called neuroses, declares that his neurasthenic patient, who had distressing delusions and had even threatened suicide, was not to be regarded as a case of certifiable insanity, there need be little surprise that the attachment of a diagnostic label proved insufficient to prevent the case from shortly afterwards committing *felo de se*.

Again an inspector of poor recently telephoned to say that a woman had attempted to gas herself and had cut both wrists with a razor, but the doctor could not certify her as insane. I agreed to the proposal of the inspector to bring the patient, meantime arranging for two medical men to attend at the asylum, where she was duly certified and admitted.

Certificate writing is becoming a lost art, since the voluntary system has become so fashionable. It is not uncommon now to have a doctor put through an urgent telephone call requesting advice as to the disposal of a patient. He confesses to an entire ignorance of procedure and inability to frame a certificate of insanity. On interrogation it may transpire that the patient is depressed, agitated, and expresses a desire to commit suicide, or is creating a disturbance, violently ejecting relatives from the house, and in one case even menacing them with an axe. Having extracted enough facts indicating insanity I suggest a form of certificate for the doctor's guidance and the patient is duly admitted, with a rather mutilated version of it appearing in the admission papers, and a passable copy of this inscribed and signed by another practitioner as certificate No. 2.

The Padded Room.

It is particularly unfortunate that the General Board in their Annual Report for 1935 should, in a praiseworthy but misguided if not misguiding attempt to soothe the public, make the statement that "the disuse of the padded cell is among the more evident improvements which have taken place in modern asylum care and treatment". While it is true that this unsightly term is of common usage among the general public, it is equally certain that no member of a mental hospital staff would so describe this special type of apartment.

I am utterly opposed to the view, which promptly appeared under glaring captions in a widely read newspaper (quoting this report), that this method of treating some of the most anxious and difficult cases of mental illness should be regarded as obsolete. Our single padded room is often unoccupied for long periods—even nine months at a time—but there are occasions when were

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three or four such rooms available, they would prove the greatest boon to our patients and nursing staff, as regards the safety and welfare of the former and the anxious duties of the latter. Many a case of acutely delirious excitement in an extremely weak physical state, liable to stumble or fall out of bed and sustain serious injury by coming in contact with the hard floor of the ward, and with whom it would be dangerous to engage in an unseemly and exhausting struggle, or to incur the risk of a seriously lowered vitality being further reduced by the administration of sedative drugs, is tided over the critical phase of his illness to be ultimately discharged recovered. There is no question in my mind but that the padded room thus judiciously employed is the most humane and efficacious method of dealing with such cases, in that it provides the irreducible minimum of risk of the patient's collapse and death.

I know that this view does not receive the support of some of my colleagues who pride themselves in having dispensed with the padded room in their institutions! It is rather amusing, however, to learn that in a recent nursing examination of this Association, not a few of the candidates in describing the nursing of a case of general paralysis gave a detailed account of what was virtually an improvised padded room. This was attained by completely covering the floor and lining the walls of a single room with mattresses. It would appear that in those places where the padded room has been abolished, or not provided, in deference to the opinion of some of our more modern psychiatrists, it is still found necessary to teach the nursing staff how to get over the difficulty. The padded cell ceases to be a cell when it is a padded room. But does a padded room cease to be a padded room when the lining consists of mattresses?

Again the view, so often expressed in various publications, popular press and platform speeches, that "living behind locked doors" is the unenviable lot of the mental case, is an unjust reflection on both patient and asylum care, which only serves to foster public prejudice. Only in the rarest instances is there any real need for locked doors could the ratepayer be induced to provide the hospital with a larger staff. The locked door is to a great extent a matter of convenience and economy in the absence of a sufficient number of nurses to ensure the necessary supervision of those patients who are apt to stray into other wards. It seems scarcely necessary to observe that there are other modes of egress than the doors available in any ward from which a patient is determined to escape.

Calculated on the normal accommodation at Glengall, it will be found that 55% of the men and 60% of the women are resident in wards the doors of which may remain open till dusk. And in the absence of boundary walls or railings, fenced airing courts or gardens with impenetrable camouflaged hedges in our modern mental hospitals, surely it is a harsh and senseless disparagement to describe the lot of the mental patient as one of "incarceration"!

In this connection I well remember how, forty years ago, Clouston held in

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utter abhorrence the use of such terms as "incarceration," "refractory ward" and "airing court," and his never-to-be-forgotten dictum that "asylums from which there were no escapes were improperly conducted".

Restraint in Nursing Homes.

To-day there are quite a number of homes, hospitals great and small which are solely or in part catering for the treatment of so-called minor mental ailments or "nerve cases"; where patients are being subjected to a form of treatment that in mental hospitals requires to be recorded in a register and reported to the General Board of Control. I refer to the employment of restraint, which is very seldom practised in asylums nowadays (I have tried it once only, and that forty years ago), but is not uncommonly employed in private houses, nursing homes and similar places without any kind of supervision or inspection by the statutory body provided for the protection of the insane by the Lunacy Act of 1857. Restraining sheets are commonly applied in cases of restless delirium in general hospitals, where no report is demanded by, and so none is given to, the General Board. In consultation at a nursing home quite recently I saw a patient who had been getting hypodermic injections twice daily for several weeks to keep him quiet and put a stop to his continuous and disturbing endeavour to get out of bed. On turning down the bed-clothes I discovered the cause of his frantic efforts. He was tied by the ankles to the bedstead!

This man was promptly certified, and after a drugless sojourn in the padded room for a few days, with a complete avoidance of struggling of any kind, made an uninterrupted recovery.

Malarial Treatment of General Paralysis.

In a lengthy circular the Board say they are not disposed to interfere generally with regard to the adoption by any medical superintendent of experimental treatment so long as the written consent of the patient's nearest relative has been obtained. They go on to say that the direct method of inoculation is open to serious objection and recommend the proper strain, where it may be procured, and explain how payment is to be made to the Board, including travelling expenses and subsistence allowance and cost of time incurred by the Ministry of Health officers in securing the particular strain required. Yet the Board deplore the under-staffing of asylums, which statement in an annual report is promptly seized upon by a newspaper, which produces a leaderette headed Neglect and ending with the alarming assertion that "the insane are still shockingly neglected". The Board further desire to be informed by medical superintendents of every case in which treatment is given as well as of any curative results. Surely a departure from an indisposition to interfere, expressed in the first paragraph! But the final instruction should still less serve to inspire the enthusiasm even of a budding scientist.

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In it the Board add that in the event of a patient dying in an asylum (note the residence) as the result, either directly or indirectly, of being infected by the malarial parasite, the occurrence should be fully reported to the Board and also to the fiscal. Can you wonder at my disinclination to take any such risk? Can you picture the dilemma of the Procurator Fiscal when requested by a doctor to have himself arrested on a charge of manslaughter, the result of experiment instigated by a Government department?

Divorce for "Incurable" Insanity.

Since the Majority Report of the Royal Commission, twenty-five years ago, considerable changes in social habits have taken place, but none so unfortunate as that resulting from the doubtful security afforded the public in the provisions for the treatment of venereal disease.

The Report says, "We have given anxious consideration to this important subject, and are satisfied that it will be in the interests of the parties affected by cases of lunacy, to the interest of their children and of the state and morality, that insanity should be introduced as a ground for divorce". And again, "The sane spouse is unable to add healthy legitimate children to the State and continue the family name. The poor man whose wife is in an asylum for life must find a housekeeper to manage his home and take care of his children" —who apparently never grow up!

A writer to the Press recently makes this statement: "It is well known that in a large percentage of cases insanity has been caused by the irregular or vicious habits of the insane partner . . ." Again—"And yet for these diseased persons we are to sacrifice the interests, not merely of the sane married persons, but of the children of the future", and "on eugenic grounds alone the proposed legislation should be welcomed."

A few points in illustration of the other side of the picture may be cited from my own experience of over forty years as an asylum physician in English and Scotch, private and public institutions. First, I assert with the utmost confidence that far more cases of insanity are caused by the irregular and vicious habits, not of the insane person, but of the spouse. Where before the war it was a rare thing to admit a woman who had been deserted, or who had obtained a separation or divorce from her husband, in recent years this unsatisfactory state of affairs has been increasingly common. There is often a history in cases of newly admitted women patients that the husband "has gone off with another woman", and the mental attack was to be ascribed to the wife's unhappy married life. In such cases the insane wife is deprived of all incentive to recover and she sinks into an incurable melancholia, because the place in her home has been usurped by the woman whom she had long suspected of having stolen the husband's affections. It is quite common nowadays to have an authentic history ascribing the wife's mental illness to neglect, abuse

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and ill-treatment by her husband, and it is particularly in most cases of general paralysis that the wives owe their syphilitic infection to their husbands' misconduct. The fact that her children are the innocent victims also of their father's immorality is scarcely a matter for pity to be extended to him. That such a man, on account of his wife's incurable insanity, which her chances of obtaining divorce precludes, should be allowed to re-marry to infect another wife is hardly a form of justice that could make any appeal even to the most ardent advocate of any Divorce Bill! Again what can possibly be said in favour of the husband of two wives, successively admitted, but in whose cases the intensive treatment so optimistically applied had been ineffectual, while the real offender has been lucky enough to have a remission of the symptoms of general paralysis? More than thirty years ago I admitted to the asylum within a few months of each other a married woman and the married man with whom she had had adulterous relations, to be followed later by her husband and the wife of the other man—all four suffering from general paralysis, from which they ultimately died.

During the course of my professional life I have seen many cases of a non-progressive form of the disease in the husband who regularly visited his insane non-resistant victim till she died in due course. In not a few cases it is not until the husband has been admitted to the asylum in an advanced stage of general paralysis that the wife has been afforded an opportunity to have herself and children subjected to a blood test, only to receive the report that all the family are infected.

In spite of the vast amount of literature perused on the subject I am convinced that no one who has been infected with syphilis ever gets rid of it. Be the period of latency short or long, it is only a matter of time till the nervous system, chiefly the brain, becomes the site of the inflammatory and degenerative changes which ultimately end in death, provided the patient is not carried off by some intercurrent affection before the too obvious signs of these changes present themselves.

More than thirty years ago Prof. Wyllie remarked to me that he saw ten times more cases of general paralysis than Clouston met with in all his consulting and asylum practice. An astounding assertion, which, however, may serve to explain why the term "general paralysis of the insane" has persisted in lunacy nomenclature, since only those few cases that showed severe mental involvement came under the notice of alienists or asylum physicians, while nine-tenths of the incidence of this disease went unrecognized by the relatives or general practitioner. Being a disease that is notoriously non-progressive or stationary for long periods, the patient may ultimately have a fatal cerebral seizure or die from a rapidly developing congestion of the lungs. In illustration of such cases being missed, I would cite that of a young man whom I lately saw in consultation. I had no hesitation in diagnosing the case as one of general paralysis, and even told the doctor so, but as his mental attack was subsiding, the relatives

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decided to delay sending him to the asylum. A few weeks later, happening to meet the doctor I inquired about his patient, to get the ingenuous reply: "Oh, he died of pneumonia in the county hospital last week"! And it is from such data in death certificates that the Registrar-General compiles his statistics as to the prevalence of general paralysis in the community!

It is not uncommon, in my experience, to have a case sent to the asylum with the diagnosis of epilepsy, which to the trained mental nurse, to say nothing of the physician, is obviously one of general paralysis. Here again statistical returns for epilepsy are apt to be vitiated by including undiagnosed cases of general paralysis.

When I read reports about the unsatisfactory control of venereal disease, and particularly in the case of one very large city where 60% of the patients receiving treatment at various public clinics fail to attend in the course of a few weeks, I feel that it scarcely requires mere scepticism as to the cure of even thoroughly treated cases of syphilis or general paralysis to support the view that there is an appalling amount of syphilitic disease in the country. That there is good reason for alarm is only too apparent when the results of efficacious treatment and how it is to be obtained are recorded for our perusal by one of the foremost experts of the day. He describes in a recent paper how in a series of his cases the patients were subjected to simultaneous intravenous and intramuscular injections of differing substances, repeated at intervals for a period of months, to be followed by courses of treatment with various special preparations, extending over several years, during which periodic examinations of both blood and cerebro-spinal fluid are made. He claims that a complete cure is assured to permit of its being safe in the interests of the offspring for the patient to marry ten years after incurring the infection!

That the lack of self-control in an individual should necessitate such prolonged and arduous treatment and skill is deplorable, however much one must admire the devotion of the scientific and humane investigator. It would appear therefore, when all is said and done, that a higher standard of citizenship on the part of every individual is the only hope for the eradication of this, the greatest of all social evils, which continues to wreck the health and happiness of countless thousands.

Psycho-analysis.

In an appendix to his book on *Psycho-analysis and Social Psychology*, McDougall gives as his excuse in finding arguments for the rejection of Freudian errors the evil social consequences that have resulted from them.

He holds that Freudian teaching has had, and is still exercising most destructive effects throughout our Western civilization, destructive both of the happiness of a multitude of individuals and of the moral traditions of society. Seriously destructive of happiness and sound tradition has been the pan-sexual doctrine—especially the doctrine of the Œdipus complex.

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This has worked in two ways to spoil the relations between parents and children, relations which are so essential to the flourishing of the family and of all our culture and civilization. Parents have been made fearful of manifesting the least affection for their children. They have been taught that all such manifestations are libidinous, are sexual displays which inevitably provoke sexual responses from their children and promote the formation of the dread Œdipus complex. On the other hand, young people have been taught that their chief moral duty, essential for their attainment of maturity, is strict avoidance of all manifestations of respect, affection, sympathy or esteem for parents.

McDougall, even if convinced of the truth of the pan-sexual doctrine, still rates highly the moral worth and beauty of filial devotion. He considers that the relations between the generations are already endangered by the many violent changes of the social order which we owe to physical science. But Freudian psychology has worked as an additional disruptive force, especially among the classes which have the function of moulding social tradition and practice. The need for this alarm is suggested in the statement by Hamblin Smith, where he asserts that "the real value of Freud's great hypothesis will be found in the school and in the home". He, however, does not claim, he says, psycho-analysis as a short and lazy road to the rectification of all social evils.

There can be no doubt, says McDougall, that the errors of the Freudian psychology, with its various popular misrepresentations, have vastly promoted the over-emphasis on sex, the importance of sex expression and sex relations of all kinds. Practically, Freud's teaching, filtered and distorted in many ways, works upon the multitude as a precept against all restraint in sex matters. He seems to say to the man in the street: "You are all sex. Whatever you do is sexually motivated. . . . Be frankly sexual and you will be both happy and healthy. Restrain your sex impulse at your peril; that way lies neurotic disorder and insanity." This, of course, was a well-known medical and popular view many years ago which was combated convincingly by Acton in his book on the reproductive functions. He was ignored, if ever even known, by a certain school of medical practitioners, as I learnt by experience in examining the nervous wrecks while acting with the deceptive title "neurological specialist" on Ministry of Pensions Boards after the Great War. It is interesting to recollect that a wholesome talk with these patients on the fallacy of the prescription of incontinence effected a rapid improvement in their morbid mental state.

Contraception and the Population Problem.

I am strongly convinced that the use of contraception is an interference with a natural process and that Nature will not be so flouted with impunity. The day comes when morally, mentally or physically the offence will have to be paid for.

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It is undoubtedly true that economic and other conditions of modern civilization have been rendering it extremely difficult for parents to bring up more than the barest minimum of children. But surely to match this evil by the introduction of another evil is no way to cope with the mischief. Even the time-honoured expedient of choosing the lesser of two evils has not the remotest chance of even partial success in overcoming the difficulties of such a serious national predicament. In my own view it can never properly be considered the province of a member of the medical profession to devise or employ methods for limiting procreation among the general community on the grounds of social economy. There is no evading the fact that the decline in the birth-rate of this country is now a matter of the gravest import, and is very seriously exercising the minds of all thinking people.

The whole problem of contraception must have the most earnest consideration of all sorts and conditions of men and women who desire the betterment of mankind, mentally, morally and physically. As the remediable causes are already well known, there need be no great delay in devising new methods and alterations in conditions of living to overcome this social blight, nor need there be any waste of time in seeking statistical evidence before getting to work.

In my view and that of many others, there is still plenty of room on the earth's surface and there are ample resources to permit of a considerable increase of the human race if only man had the wit to exploit them. We have in recent years heard so much of the evils of speed, but little attention is given to the evils of size, viz., the massing of the population in cities, which so often proved the downfall and decay of past civilizations. Modern science is progressively providing rapid transport and inter-communication, and with amusement and culture by means of wireless available in every dwelling, surely man will have ample opportunity to return to the joys of family life. Speed, instead of being an evil, should, if properly used, prove a means of allowing the peoples of the earth to spread and thus make room for everybody.

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THE CIRCULATORY SYSTEM AND MENTAL DISORDER

A SYMPOSIUM HELD AT THE ANNUAL MEETING OF THE ROYAL MEDICO-PSYCHOLOGICAL ASSOCIATION AT AYR, JULY 8TH AND 9TH, 1937.

INTRODUCTION: SOME REMARKS ON THE PHYSIOLOGY OF THE CEREBRAL CIRCULATION.

By F. L. GOLLA, F.R.C.P.

Prof. GOLLA, in introducing the discussion, said: I do not think there is any subject that is of greater importance at the present moment than the physiology and the pathology of the circulation of the central nervous system. I am dealing now with the physiology, with a view to putting a few facts before you which are fairly well authenticated, and pointing out how very little we know at present. I do not intend to speak of the pathological aspect, but I may remind you, in passing, of such things as the therapeutic effects of amytal and of CO_2. Again, in the case of epilepsy, whatever one's ultimate view of epilepsy is, we are all agreed that circulatory disturbance plays a very large part.

Now for a few facts which make it a little clearer why it is that disturbance of the circulation of the brain is likely to be so very important. The need of the nerve-cell for oxygen conveyed by the blood is overwhelming when compared with the similar need of any other tissue in the body. The brain, in man, represents 2% of the body-weight, but the brain accounts for 10% of the total oxygen consumption of the body. The cortex consumes about ten times the oxygen that is consumed by the white matter, and when we speak of the cortex we must remember that the nerve-cells, which are chiefly responsible for oxygen consumption, represent only 1% to 2% of the total volume of the brain. Unlike any other tissue, the brain cannot establish an oxygen debt; it must be continually supplied with oxygen by the circulation.

The amount of blood flowing to the brain is a very difficult thing to determine even approximately, because, as those who deal with the anatomy will show, there are such a number of possible afferent paths for the arterial blood. But, basing one's figures on some work which was done by Gazda a good many years ago, 100 grm. of brain probably receive 140 grm. of blood per minute. In other words, the blood-supply per minute of the human brain is about 1½ litres. So the one thing to notice is that the mechanism is so arranged that, whatever else happens to the body, under no circumstances shall the brain be deprived of its quota of blood.

We can look at these circulatory problems from two points of view: Firstly, how is the blood-supply of the brain maintained in spite of the alterations in

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posture and in blood-pressure? Secondly, and what is much more interesting to us as psychiatrists, is there any mechanism by which additional activity of different portions of the brain, can be provided for by additional blood-flow to that particular portion?

I think we had better deal with the second of those problems first, because it is very much more important to us, and it is one in which phantasy can very easily play a great part, because, unfortunately, our physiological methods are very deficient when we talk of the circulatory activity of the brain. Have we reason to think that the metabolism of the cortex increases in response to increased intellectual activity or increased sensory stimulation? There are two ways in which one can show that this does happen. One—the better—is to use the temperature of the brain in its different parts as a check on the amount of blood flowing to that part, and this is done by means of thermo-electric couples inserted into the brain. One finds, as one would expect, that excitatory drugs, such as strychnine, will increase the temperature. Pain also will increase the temperature, and the mere exhibition of light to a curarized animal will cause an increase of temperature, which, according to some, is confined to the occipital lobes; I feel doubt about that, however. If we attempt to find out the oxygen consumption of a stimulated brain or an inhibited brain we get enormous variations. The figures are only approximate, because our methods are defective, but, roughly, an animal undergoing strychnine convulsions consumes 700% more oxygen than does a resting animal; with caffeine it is 150% more, with cocaine 120%. In the animal poisoned to the extent of complete anæsthesia with luminal, the oxygen consumption is reduced by about 20%; with other barbiturates the oxygen consumption is diminished by 30% to 40%. These two facts, taken together, show that fluctuations must take place in the blood-supply of the brain in order to account for this huge increase or decrease of oxygen consumption; and the temperature observations with the thermo-electric couple confirm that.

What is the mechanism by which such things can happen? At first it might be thought possible to get an answer by measuring the flow of blood to and from the brain. Unfortunately, there is anastomosis between the carotids, and from the arterial point of view there are other difficulties, because one has to consider the blood coming by the vertebral arteries, etc. Similar difficulties are in the way of the estimation of the amount of venous blood. Therefore our figures have been, to a great extent, approximate.

I must now go back to the question of oxygen consumption. Would it be possible to show that different parts of the brain consume different amounts of oxygen? All we know is that cortex consumes about six times as much oxygen as does white matter, and that if the oxygen consumption of portions of the brain, taken separately and enclosed in a bottle and kept at a uniform temperature, is measured, it is found that the consumption in different parts of the brain varies as the vascularity. These variations seem to depend on the synapses present. The nerve-cell itself, if we judge by experiments on the posterior root ganglia, which have no synapses, has a low oxygen consumption. With the synapses, however, there is a great increase in oxygen consumption. Marinesco's results seem to be confirmed, namely, that the chief site of metabolism is at the synapse.

What means have we of altering the blood-supply to the brain under normal conditions? One would expect, as in the case of any other vascular system, that we should be able to cause dilatation and contraction of the blood-vessels, and many experiments have centred round an attempt to show

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that one can cause, by nervous influence, a contraction of the cerebral blood vessels. About dilatation of the blood-vessels we know something. The vessels of the brain are supplied by myelinated and unmyelinated nerve-fibres, forming part of the parasympathetic system, and stimulation of the vagal system causes dilatation of the blood-vessels. But when we come to constriction it is a different story. Our methods are such that it is very difficult to be certain that any contraction of vessels takes place in response to any form of nervous stimulation. If the cortex in an animal is exposed, electrical stimulation will cause immediate blanching, which may last four or five minutes, sometimes even as long as two hours after a single stimulation. But that, I think, is a purely local phenomenon, and if we search for what we are expecting—the sympathetic nerve supply acting in opposition to the parasympathetic—that is to say, if we look for vaso-constrictor sympathetic fibres, we do not find them. Probably the only really valid method which we have at present is one we owe to Risi and his French co-workers. It consists in trephining and painting the dura with coagulin. It then becomes transparent, and one can observe pial vessels and take photographs. Under these conditions, stimulation of the cervical sympathetic has no effect on the cortex or on the vessels of the brain. One finds after injection of drugs such as adrenaline that this has no effect on the calibre of these vessels. Previously we had accepted the statement that there were proofs that adrenaline caused vaso-constriction, but now we can be sure that nothing of the sort happens. No experiment has been devised that will show any vasomotor mechanism to cause constriction of blood-vessels of the brain. Histologists tell us that they can see short fibres along nearly all the blood-vessels, but they agree that it is difficult to be certain about this. They always fail to trace them for more than a short distance. These fibres appear to be analogous with Auerbach's plexus, and to have no direct connection with nerves coming from outside. So there is the curious fact that we have a mechanism for the dilatation of the vessels of the brain, but none for their constriction. I suppose the real answer is that we do not particularly need it. It may be that the needs of the central nervous system are conveyed chemically, and not by nerve impulses. There are certain facts which seem to point to the possibility that in the central nervous system we have a circulation of chemical bodies. Time-relations have to be short, and when one deals with these molecular layers, chemical reaction can be infinitely short. Consider what happens in the castrated animal, that is, in an animal castrated from infancy, the external genitalia being removed. If such an animal is injected with male hormone it begins to make attempts, which are abortive, to imitate male sexual movements; and this cannot be due to turgescence, since the genitalia have been removed. Presumably the hormone acts as a specific on the central nervous system. Similar events may be happening all over the central nervous system. There are many facts which seem to favour the idea. It may be that the local vaso-constrictor mechanism, which we know to exist, and which we can stimulate locally, is one which is aroused by various internally conditioned hormones, not necessarily the ones secreted by the endocrine glands. We have already got an insight into the possibility of secretion of specific bodies by nerve-cells themselves; they may act as their own hormone producers.

So we are in this unsatisfactory state: We know about dilatation, we know of drugs which dilate, but we do not know of drugs which constrict when injected intravenously. The chief dilator of interest in psychiatry is CO_2. This explains the results obtained in the treatment of schizophrenia

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by means of CO_2, and the dramatic results of the administration of amytal. CO_2 possesses the great property of dilating the blood-vessels of the nervous system, although it constricts them elsewhere. It is a mechanism by which the organism protects itself against accumulation of CO_2 by increasing the circulation.

The other substance of importance is caffeine, which is a general stimulant to the cortex; it causes dilatation of the blood-vessels of the cortex. There are not many other substances of which one could say without doubt that they cause vaso-dilatation. Ether is the only one of importance, unless we include histamine. Histamine is interesting because from it we can learn one or two physiological facts, and a few things which interest us as psychologists. It causes a fall of blood-pressure and a dilatation of the cerebral vessels. Dilatation of cerebral vessels hangs a little behind in the process of recovery from the fall of the blood-pressure when a person or animal is poisoned with histamine. Now persons who have had an injection of histamine suffer from intense headache some time after, lasting for a minute or two. It has been shown that the cerebral vessels remain dilated for a minute or two after the general blood-pressure has gone up again, and in that time there is such an increased flow of blood to the membrane vessels that they become distended, and this causes the short intense headache.

Had there been time one might have dealt with our protective mechanisms against alterations of posture producing bad effects. I do not know in how far they necessarily interest us as psychiatrists, but I think they perhaps do so more than one is inclined to think. Some years ago I carried out some elementary observations on schizophrenics, and I found that the readjustment of blood volume in those people when rising from the stooping to the upright posture, and vice versa, takes place more slowly and less efficiently than in the normal person. The need for an efficient mechanism is obvious when you think of the case of the giraffe, and reflect on the difference there is when it puts its head into the trough and then raises it in the air; yet apparently that animal does not suffer from symptoms in consequence.

Relative vascularity of the brain we know a good deal about as a result of injection experiments and the beautiful preparations which Dr. Pickworth is showing us, which give an indication of the vascularity of an area of the brain, not by staining the blood-vessels, but by staining the blood which is in them. If only we receive these results with caution and apply them only where they should be applied, I think Dr. Pickworth has given us a weapon which will be of the greatest value. But I would remind Dr. Pickworth that people do not die of insanity—one wishes sometimes that they did; they die of all sorts of other diseases, and death is a very big factor. It is a very important matter for a person to die; there are respiratory and cardiac factors. During the last hours of agony events are taking place which are more important than the derangement of conduct which we call insanity. So I feel a little doubt whether one will be able to find, by any histological preparation, that a particular brain has been more or less well supplied with blood during the course of the mental illness. However, to that question Dr. Pickworth will probably be able to give us an answer.

I may perhaps have been able to convey to you the idea that we know exceedingly little about this subject, and that it requires more thought, but that it may prove to be of greater importance, especially as to its relation to disturbances of the nervous system, than possibly any other subject at the present moment.

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THE HISTOPATHOLOGICAL CRITERIA OF VASCULAR DISTURBANCES IN THE BRAIN.*

By ALFRED MEYER, M.D.Bonn.

[Abstract.]

VASCULAR lesions, identical in appearance with the sequelæ of organic diseases of the blood-vessels, have been found in a great variety of conditions (for instance in epilepsy, hypertension and cardiac disease, psychoses associated with infectious and toxic conditions, head injuries, certain groups of mental deficiency) in which no organic lesion of the blood-vessels themselves could be demonstrated. The theory has been put forward (Ricker, Spielmeyer and others) that these lesions owe their origin to functional disorder of the vascular system, and that this common factor accounts for the identity in histological appearance in spite of the profound ætiological differences.

In this paper the material is surveyed with a view to laying down the criteria for the histological diagnosis of vascular lesions. Certain regions of the brain which are particularly vulnerable serve as indicators in the pathogenic analysis. An attempt is also made to define more clearly the nature of the functional disorder responsible for the lesions.

* The full text of this communication will be published in a later number of the Journal.

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THE RELATIVE VASCULARITY OF DIFFERENT AREAS OF THE MAMMALIAN BRAIN.

By A. COLIN P. CAMPBELL, M.B., Ch.B., M.R.C.P.Ed.,
Pathologist, Scottish Asylums Laboratory.

[*Abstract.*]

THOUGH many of the earlier workers made statements based on general impressions from injected preparations about the relative vascularity of different parts of the brain, only comparatively recently has an attempt been made to measure and express numerically the capillary development in different areas, by methods similar to those used by Krogh in studying the vascularity of muscle. Craigie has made an extensive study of the rat brain by such methods, Cobb, and Cobb and Talbot have investigated certain areas of the rabbit brain, and Dunning and Wolf certain parts of the nervous system of the cat. In the present paper a personal investigation (undertaken at the suggestion of Dr. Cobb) is reported, comparing the basal ganglia, cerebral and cerebellar cortex of the cat as regards capillary vascularity. The capillaries were demonstrated by Indian ink injection, and results expressed in millimetres of capillary per cubic millimetre of tissue. Substantiating observations were made on similar areas of monkey and human brains, but without attempting accurate measurement as in the cat material.

The results indicate a striking variation in vascularity in the various areas of grey matter examined; the globus pallidus was found to be by far the least vascular area; the basal ganglia were on the whole less vascular than cerebral neocortex or cerebellar cortex, though the lateral geniculate body had as high a vascularity.

To a certain extent Craigie's statement that the receptive and associative areas have a higher vascularity than the efferent areas was confirmed. Furthermore, a correlation could be made between the vascularity and the richness in ganglion cells of the various areas; there were, however, striking exceptions to this correlation—for instance, the granular layer of the cerebellum has a relatively low vascularity in view of its extremely dense cellularity. This, like Dunning and Wolf's demonstration of the relatively low vascularity of the cat's trigeminal ganglion, suggests that the metabolic activity of an area of nervous tissue (as measured by its vascularity) is not necessarily in proportion to its richness in ganglion cell bodies.

The relationship, if any, of richness or poverty of capillary development to

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the selective localization of anoxæmic lesions remains obscure. That selective necrosis in carbon monoxide poisoning occurs in the globus pallidus, the least vascular area of grey matter so far examined, is superficially suggestive; but while the vascularity of this area is poor, so also may be its oxygen need for survival (as is suggested by its sparseness in ganglion cells), and there is no means of knowing as yet whether this or any other area has a low margin between anatomically available capillary bed and oxygen need.

Discussion.

Dr. A. MEYER asked whether there was any difference in the cornu ammonis, i.e., the vulnerable sector of it, as compared with the non-vulnerable. He had never contended that the smaller number of capillaries in the globus pallidus was the cause of its vulnerability. Hiller had expressed that view, but the speaker had not adopted it.

Dr. CAMPBELL replied that he was sorry if he had misinterpreted Dr. Meyer's opinions. In reply to Dr. Meyer's question, he, the speaker, did not know whether the non-vulnerable sector showed a different vascularity; he had not done the counts in the cornu ammonis. He took thick sections, and gained impressions from them. He hoped to measure the vascularity in different parts of the cortex.

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CEREBRAL ISCHÆMIA AND MENTAL DISORDER.*

By F. A. PICKWORTH, M.B., B.S., B.Sc., A.I.C.(Exam.),
Director, Joint Board of Research for Mental Disease, City and University of Birmingham.

IT has been calculated that over one-third of the total number of cases receiving sickness benefit have some form of nervous disorder (1), and 1 of 22 persons has to be placed in a mental hospital at one time or another (Carrel, 2). Social prejudice, however, still bars adequate support of psychiatric research, and much scientific work is hampered through lack of financial help.

Focal cerebral anoxæmia in relation to the ætiology of mental disorder has occupied my attention for many years, forming in 1929 the subject of my contribution to the Mott Memorial Volume (3). My efforts were then limited to the collection of evidence of neuronic changes caused by oxygen deficiency, but the correlation with the clinical manifestations of psychiatry presented difficulties. The prevailing opinion of my teachers and colleagues, as to the ætiological causes of mental disorder, was that of biogenetically deteriorated or toxin-impaired cerebral neurones: visualized as having swollen or sclerosed cell outlines and processes, containing fatty globules or pigment, nevertheless making sporadic but futile attempts to carry out function, with the inevitable result of a disordered mind. The fundamental obstacle to this theory is the occurrence, in some cases, of natural remissions and of dramatic restorations of sanity during acute infections (e.g. pneumonia), or just before death: also excitation of structurally disordered or toxin-impaired neurones to be in consonance with the theory, should produce even more bizarre mental effects than before, whereas actually tranquil mental order and restoration of disintegrated personality are clinically observed. A few months later Loevenhart (4) demonstrated restorations of sanity by his methods of cerebral stimulation, giving further proof that not only the neurones but the neuronic patterns may be intact and capable of functioning after years of inactivity and disorder. Other methods of cerebral stimulation are now known, the most important of which, in connection with the present paper, is that of Hallay (5), who used "massage of the brain", which consisted of alternate compression of the jugular veins. The biogenetic theories of deteriorated and toxin-impaired neurones which we have cherished so long thus become

* Read at the Annual Meeting of the Royal Medico-Psychological Association, July 8, 1937.

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untenable, and the colossal literature on the subject remains to indicate the time and energy expended in that direction.

The two main characteristics of mental disorder are mental dysfunction and a limitation of mental development with age, forming the two clinical groups of psychiatry and mental defect, but nevertheless by no means confined to these groups. In general, mental disorder presents wide differences from well-known acute or chronic bacterial and virus diseases, so that any simple infectious agent as a cause of the clinical condition is quite unlikely. In a few cases (such as syphilis, sepsis, lethargic encephalitis and rabies) where mental disorder has an intimate association with a specific infection, it is obvious that another factor of dominating importance must be present. For example, the spirochæte of syphilis may be present in the brain within a few weeks of primary infection—long before symptoms of general paralysis are due—and therefore it is not by itself the cause of the mental condition.

Much has been published on the action of toxins upon the cerebral neurones, but, as already shown, the neuronic patterns in mental disorder may be intact; also, after restoration of sanity by cerebral stimulation, a rapid relapse to the identical disordered mental state occurs, making even a theory of reversible toxic action inadmissible. A similar non-toxic phenomenon is seen in senile but otherwise mentally normal acquaintances: when undisturbed, speech and movement are slow, the eyes are lustreless and there is lack of expression; but mention a favourite topic and gradually the facial furrows disappear, the eyes sparkle, the features show emotion and there is an associated mental acuity; on changing to another topic of little interest the whole is reversed, like a cine-film put through the projector backwards. In the first few days following admission to a mental hospital the patient's mental state may be temporarily so improved that certification is sometimes difficult, and the well-known temporary improvement following any new treatment in mental disorder cases has given a bias not only to the multitudinous forms of treatment attempted in mental hospitals, but also to patients and their relatives in the use of much-vaunted patent medicines and palliative home treatments which are quite devoid of any real therapeutic value.

With possibly a few exceptions, mental disorder cannot be considered as due to any germ-plasm defect. Tuberculosis and general paralysis were until recently considered hereditary, and the work of Paterson (6), Penrose (7), Findlay (8) and Watson-Williams (9) has shaken the evidence upon which some of the former hereditary theories were based.

Importance of Circulatory Changes.

The inseparable association of nervous mechanisms with motor behaviour has obscured the fact that mental and emotional states are something apart from conditioned reflex action. A reflex is instantaneous in origin and effect,

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in contrast to the prolonged duration of mental and emotional phenomena ; this in itself is proof of the necessity for another physiological mechanism pertaining to such states distinct from the central nervous system, a proper understanding of which is essential to the study of the physiology of the mind and of the pathology of mental disorder itself.

Mott (10), Hill (11), Spielmeyer (12) and others recognized the hopefulness of study of the vascular supply of the brain in relation to the mind, but they found the extreme vulnerability of the ganglion cells of the brain to oxygen lack a stumbling-block to further progress in this direction. As I have shown, injury of the ganglion cells themselves is an unlikely cause of chronic mental dysfunction, and moreover, according to Fremont-Smith (13), each ganglion cell has a separate blood-supply to itself. It is known that posterior root ganglia (where cells are numerous) have a relatively poor blood-supply, whilst laminæ III and IV of the cortex (richest in synapses) have the richest blood-supply ; lamina I, rich in synapses, has a good blood-supply, not only from its contained capillaries, but from contact with the highly vascular pia, where the colour difference between arterial supply and venous return is normally very remarkable ; the enormous amount of oxygen normally consumed by the brain therefore must be utilized at the synapses. Decrement occurs at, and only at, the synapses, and it is now known that a slight ischæmia increases this natural decrement of the synapse to extinction of nerve impulse. My work with the capillary stain has shown ischæmia of the synaptic layers of the cortex, and enables me to put forward a new theory of the physiology of mental effort, emotion and instinct, conveniently pictured as a definite mosaic pattern of capillary activity amongst the millions of synapses, facilitating some, inhibiting others, and thus predetermining the motor behaviour which instantly follows sensory impulses passing through the area. Such capillary patterns may be initiated or " set " by the sum total of the activities of the central nervous system, but persist owing to sympathetic nerve action upon the small arteries and arterioles, and/or the action of metabolites and hormones upon the capillaries. Potential mental and emotional states thus exist without necessarily any concomitant activity of the central nervous system, and have an anatomical representation in the patterns of capillary activity. These capillary patterns, though complex, are infinitely less complex than the neuronic arborizations and synapses whose function they control. Any sensory nervous impulse through a chain of synapses must have a fixed type of motor response, which is determined by the capillary pattern existing at the time. The work of Sherrington (14) and Pavlov (15) is thus simplified ; conditioned reflexes do not depend upon the *permanent* establishment of a fixed path through alternative synapses, but are part of the essentially *labile* state of delicately balanced alternative synaptic paths, regulated by the conditioned mosaic pattern of vascular activity. The combined possibilities of such patterns determine the personality of the individual, and pathological changes in the

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capillary patterns are believed to be a fundamental cause of perverted mental activity, abnormal behaviour, and disintegration of personality, seen at their worst in mental hospital patients.

This theory opened up a new histological approach to the study of mental disorder and new methods became necessary; the relative vascularity of local brain areas for obvious reasons could not be satisfactorily demonstrated by injection methods, and all staining methods known were unreliable for the purpose. By 1934 my capillary blood-stain method was perfected and published in the *Journal of Anatomy* (16).

As early as the year 1700 disease of the cerebral vessels was noted by Brunner. Ellis in 1909 correlated spontaneous cerebral hæmorrhages with atherosclerosis; Orr and Rows in 1917, showed that staphylococcal toxin placed against the carotid artery of animals resulted in hyaline thrombi etc., in the cerebral capillaries; and in 1928 I was able to show streptococci in the brain-vessels of an acutely excited case of dementia præcox. Jaensch studied capillary patterns, finding deformation in the brains of cretins, and Delbruch in 1927 showed that the finger capillaries of mentally defective children were mal-developed. Vogt's theory of pathoclisis based upon abiogenetic degeneration of the third layer of the cortex was disproved by Neuberger, who was able to get the same results of third-layer ischæmia in fat and air embolisms; whilst Sträussler and Koskinas found laminated and third-layer atrophy both in atherosclerosis and general paralysis. My researches show clearly that lesions in the distribution of pial arteries can be correlated with such laminated atrophy. Keschner studied the mental disorder due to cerebral tumours and concluded that symptoms were due to vascular accidents; and in 1928 von Braunmühl showed that excitement was followed by spastic hyperæmia of the cerebral vessels with capillary paralysis leading to congestion, local anæmia or hyperæmia, stasis and hæmorrhages. In 1930 Spielmeyer published an account of functional anæmia and degenerations of the cortex in mental diseases, and demonstrated that spasm of the small arteries was the cause of necrobiosis in cells of the cornu ammonis; Royle in 1932 successfully applied cervical sympathectomy to the cure of early mental disorder. In 1935 Frazer showed that sympathetic stimulation of an artery affected it locally rather than in the whole of its distribution, and Williams in 1934 showed that, on the evidence of its sympathetic nerve supply, the posterior cerebral was really a branch of the internal carotid.

The most powerful factor controlling peripheral circulation is the presence of metabolites; this, together with the blood-pressure changes effected by the sympathetic mechanisms of aortic and carotid sinus reflexes and the facial nerve, constitutes the main physiological cause of change of lumen of the cerebral capillaries. It should be noted that the arteries of the brain-stem have their sympathetic nerve-supply distinct from that of the cerebrum; also that the medial muscular coat of the cerebral arteries is deficient.

Constriction of the cerebral vessels takes place with direct electrical stimulation, which completely occludes the lumen for 15 sec. to 2½ hours after cessation of the stimulus (Wolff). It occurs when the blood becomes hypertonic, when the oxygen content is high or the carbon dioxide content is decreased, with stimulation of the cervical sympathetic, and with adrenaline, provided that the inhibition is overcome by sympathectomy or ergotamine (it has, however,

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no constrictor action on the parietal region (Schmidt)). A slight constriction usually occurs, lasting two minutes, before the dilatation due to fall in blood-pressure, however caused (Fog, 1937). Peterson says that peripheral chilling contracts the cerebral arteries as well as the peripheral vessels, and when so contracted they fail to respond to nerve stimuli. Stavraky has shown that constriction of the cerebral vessels occurs on stimulation of part of the hypothalamic region commonly known as the fear area. Peripheral capillary circulation is controlled by the Rouget cells, each of which has its sympathetic nerve-supply. Williams, however, could find no sympathetic nerves to the cerebral venous capillaries. Generally, vasoconstrictors act on both arterioles and capillaries; a dissociated action is seen in the emotional pallor of the face (capillary constriction)—often a chronic phenomenon in mental disorder cases; and in the cold blue of the extremities with constricted arterioles and dilated capillaries—common in schizophrenia.

Dilatation occurs with increased carbon dioxide (mild asphyxia), trauma, acidosis, vagus stimulation (but not when the seventh nerve is cut close to the medulla), with a temperature of 40° C. (inconstant); with stimulation of the seventh nerve; and with a fall of blood-pressure, however caused (unless vessels are already dilated, when a narrowing of the lumen may occur); with ether, histamine (a very sensitive reaction which, however, does not occur regularly if vessels are already dilated by ether), acetylcholine, amyl nitrite and adenylic acid (a product of cerebral metabolism), and carbon monoxide—which causes long-lasting maximal dilatation (Wolff). The pituitary secretion sometimes causes dilatation, depending upon conditions already acting upon the vessels; in the parietal region it has a vaso-dilator effect (Schmidt). Edinow found that ultra-violet light dilates skin blood-vessels, causing a permanent change, so that months afterwards the irradiated area can be detected by its reaction by dilatation to histamine—an important introduction to the phenomenon of latent affection of cerebral vessels. Stavraky showed that very marked pial dilatation occurs on faradic stimulation of the tuber cinereum, a similar dilatation occurring when this area is pressed upon by a small soft sponge. A dilatation could also be obtained by stimulation of the ventral and medial parts of the thalamus. Rhythmic dilatations of pial arteries (10-sec. or 2-min. intervals) were also noted. Capillary paralysis as demonstrated by von Braunmühl follows excitement. The action of CO_2 on the brain vessels contrasts with its action on the muscle vessels; in the brain its influence in causing dilatation overcomes all constricting influences, whilst in the muscle its constricting influence overcomes all dilators.

Tureen found that inflammatory reaction took place two to three days after ischæmia had damaged the tissues, the tissue becoming normal in 11 days. Hill showed that if the intra-ocular tension be artificially lowered, abdominal pressure caused intra-ocular hæmorrhage—a phenomenon which can be applied in the explanation of the origin of small cerebral hæmorrhages. The flow in the minute capillaries is remarkably constant, and according to Wolff, the vessels do not periodically appear and disappear as in other tissues. More oxygen disappears from the blood in its passage through the brain than through the extremities (Myerson and Hilloran, 1930); jugular blood is more venous than femoral vein blood (Lennox, 1931); as much as 10% of total body utilization of oxygen is consumed by the brain, and the oxygen is chiefly used up in the synapses and not in the ganglion cells (the dorsal root ganglia having no synapses, a poor blood-supply and using little oxygen; whilst lamina IV is rich in synapses, has a good blood-supply and its oxygen consumption is

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correspondingly high). The oxygen requirement of the brain tissue is so great that the difference of the bright red of the arteries and the dark blue of the veins is remarkable; the $Q\ O_2$ *in vivo* is 8·4 = 14 c.c./100 grm./minute (Himwick and Nahum), and 3 seconds is the normal circulation time of the brain as measured by radium emanation. Ten minutes' cerebral anæmia kills the ganglion-cells, and Hill and Mott showed the disappearance of Nissl substance after ligature of the carotid in cats, pointing out that the small cells were the first to die on account of the little reserve material. Too much oxygen, however, is highly detrimental to nervous tissue, which normally has a low oxidation-reduction potential; and the blood-supply of the brain is not so great as many standard text-books would have us believe—a test by my method showing it to be about one-sixteenth that of the kidney.

It was long supposed that the brain arteries were end-arteries (Cohnheim, 1872); Fay (1925) injected cerebral vessels with mercury, photographing them stereoscopically by X-rays, and his results tended to support this. Recent researches of Pfeiffer and Cobb, however, show a wealth of anastomoses in the smaller arteries; and at present it is generally assumed that there are no end-arteries in the brain. When œdema or thrombosis affects a number of anastomotic vessels the possibility of virtual end-arteries must be considered.*

The causes of that ischæmia which results in disintegration of the vascular capillary pattern are spasm, stasis, œdema, diapedesis, capillary hæmorrhages and thromboses. A limitation of blood-flow is imposed by some atherosclerotic lesions, 'aseptic' meningitis, chronic subdural hæmorrhages and cardiac decompensation, whilst hyaline changes in the vessels, perivascular cuffing and intimal thickenings reduce local respiratory exchange.

In 1927 Ricker (18) advanced his theory of prestasis, which supposed a constriction of the smaller cerebral arteries coincident with dilatation of arterioles and capillaries. Hiller added to this a possibility of irregular dilatation of some of the arterioles arising from the same constricted artery, thus diverting the already restricted blood-supply from any undilated capillaries and causing local ischæmia. The harmful effects of prestasis are partly overcome by venous obstruction, and partial compression of the jugular vein might thus become a method of treatment of mental disorder symptoms (Fig. 1). Graves's case of ligature of one internal jugular vein for mastoid complications showed an immediate mental improvement, which was maintained. Hallay (1936) produced lucid intervals in mute catatonic patients by alternate rhythmical compression of the jugular veins. The good results of treatment of obliterative vascular disease by rhythmical (2-minute) increased and decreased atmospheric pressure (19) indicate a hopeful future for mild restriction of the jugular flow in mental disorder cases. Hitzenberger warns us not to exceed a positive pressure of 20 mm. Hg. or thromboses occur, nor a negative pressure exceeding 80 mm. Hg. or hæmorrhages occur. Such secondary changes are shown by my method in brains from mental disorder

* For further recent detailed information the reader is referred to the excellent work published in a long series of papers from the Harvard Medical School, Boston.

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cases, and indicate the natural use of pressure variation as a method of compensation of disturbed capillary (mental) function.

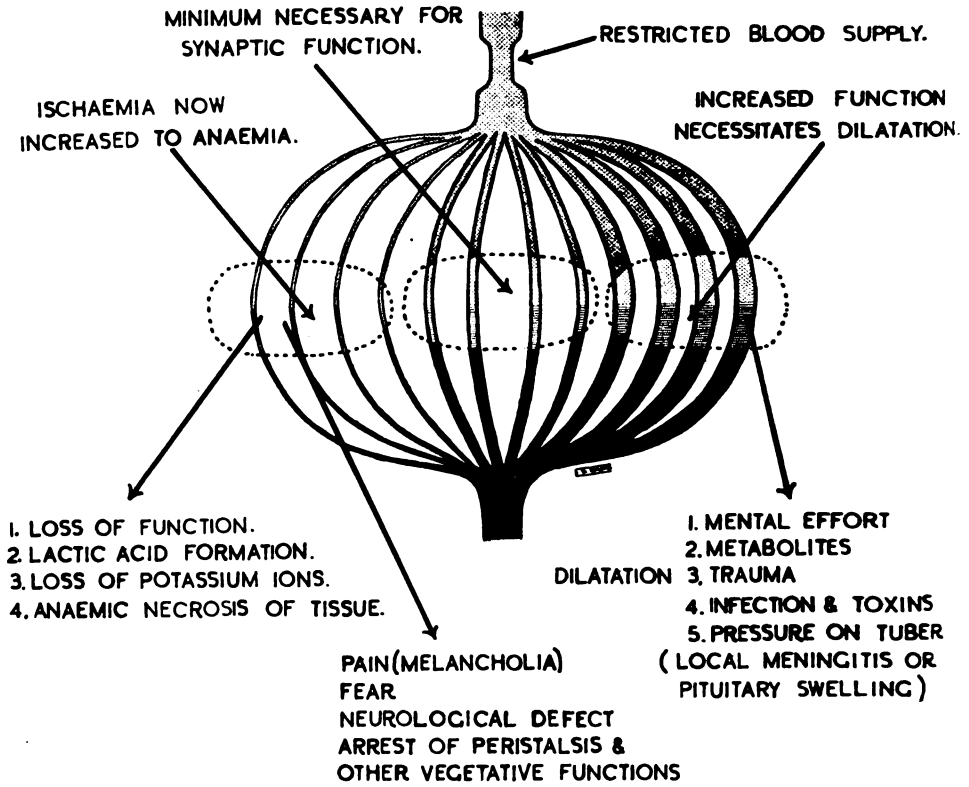

FIG. 1.—Diagram illustrating Rickers' and Hiller's theories. Centre dotted area is normal, any cause of dilatation of capillaries (right) necessitates emptying of capillaries (left) owing to restriction of total available flow. Note: Restriction of venous return would fill up the empty capillaries on left.

DESCRIPTION OF CEREBRAL PATHOLOGY.

Macroscopic.

In addition to the post-mortem findings of œdema, hæmorrhages, softenings and atrophy, I have frequently noted pale yellowish areas in the cortex and basal ganglia which are macroscopic confirmations of the ischæmia shown by the capillary stain; vascularized adhesions indicating long-past septic intracranial complications; partial occlusion of the carotid or vertebral arteries by atheroma; and partial thrombosis of cerebral arteries (which is by no means uncommon). In 1925 I collected the carotids of 50 consecutive post-mortems, finding athero-sclerotic lesions at the junction of internal and

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external carotids in every case. In my book, *Chronic Nasal Sinusitis and its Relation to Mental Disorder*, there is figured, opposite p. 32, a specimen illustrative of the long duration of traumatic disturbance of the pia arachnoid. Opposite p. 107 is shown an illustration of latent vascular disease of the cerebellum unmasked by a generalized toxin (adder-bite); it has already been mentioned that ultra-violet light causes a permanent change in the reactivity of vessels to such substances as histamine, and it is therefore changes of a similar nature to the above (which in this particular case were in all probability due to a septic complication from pulmonary tuberculosis many years previously) which are of importance in mental disorder cases. Macroscopic involvement of the carotid artery by contiguous nasal sinus infection is shown opposite p. 32, and also opposite p. 116, where the pathological changes of the artery are actually to be seen through the walls of the sphenoidal sinus. Opposite p. 116 is also figured a specimen of a sphenoidal sinus infection with thickened membrane and a thrombosis of the peri-carotid capillaries on both sides—the effect of this upon the sympathetic plexus needs no exposition. Everyone has noted athero-sclerotic lesions of the cerebral arteries; in some of these a decided restriction of blood-flow is evident, thus constituting a condition of prestasis which under certain circumstances may result in focal ischæmia. Similar lesions are seen in the vertebral and basilar arteries, where the prestatic condition in relation to the posterior cerebral artery is more obvious, since these arteries, although apparently continuations of the basilar, and only connected to the carotid by the small communicating arms of the circle of Willis, nevertheless have their nerve-supply (as Williams has shown) entirely from the carotid sympathetic plexus and not from the vertebral artery. A threatened ischæmia of the lower centres must at all costs be overcome, and thus what might be regarded as an emergency mechanism—that of restriction of blood-supply to a large portion of the cerebral cortex—becomes absolutely necessary, and must be continued permanently so long as the restricted blood-supply of vital centres is barely minimal to support function. Macroscopic pathology of the larger arteries indicates the probability of similar pathology in smaller arteries, and possible interruption of their sympathetic nerve-supply.

Microscopic.

Table I shows the number of brains examined by my capillary stain method, with the more important findings. Some work has been included in this table not satisfactorily out of the experimental stage, so that the whole must still be considered of a preliminary nature. Many more years of patient application will be necessary before the various problems which I have put forward can be settled, but this is the only method so far applicable for the working-out of the slight but chronic departures from the normal physiological

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TABLE I.—*Capillary Stain. Brains from 171 Mental Cases. (Total Sections Examined, 4439.)*

	Cases.	Is.	Cg.	Th.	Hm.	Gr.	Lv.	Pvs.
Oligophrenia (A)*	7	6	3	2	5	3	1	2
Schizophrenia (C)	18	16	14	5	9	5	2	7
Paranoia (D)	11	8	6	2	3	2	2	6
Manic-depressive: Mania (Ea) .	8	8	3	3	6	3	2	2
,, Melancholia (Eb)	7	5	6	1	4	3	3	3
,, Stupor (EaIII) .	1	1	..	..	1	..	..	..
Confusional (F)	23	18	12	3	13	7	5	3
Epileptic (G)	31	28	23	9	19	13	4	11
General paralysis (H) . . .	35	29	19	11	19	6	3	12
Organic brain disease (I) . . .	8	7	3	2	4	3	..	1
Dementia (J)	22	14	10	4	8	8	2	7

Is., ischæmia; Cg., congestion; Th., thromboses; Hm., hæmorrhages; Gr., granules; Lv., loss of vessels; Pvs., perivascular spaces.

* R.M.P.A. classification.

state which we have found to be present in cases of mental disorder. At present I am without any qualified help, but credit is due to the help of a former scholar, Dr. G. M. L. Hobart, and to my assistants, Messrs. Stewart, Rennie and Hindle for the technical application of the method.

In the normal cortex and white matter the regular structure of the vascular arborizations as revealed by my method possess a beauty of form, like that of a pine forest, which can only be appreciated by actual viewing through a binocular stereoscopic microscope (Fig. 2, normal cerebellum; and Fig. 3, normal cerebral cortex). The vessels in healthy animals are normally of even calibre and nowhere show departure from the expected regularity. In all mental cases so far examined and in animals known to be unhealthy when killed, departures from the normal regularity are found.

I have not yet sectioned a large number of brains from non-mental patients because that would not help the problem in its present stage; we are not concerned with averages, but with the absolute clinico-pathological state of undue ischæmia of cerebral tissue, and Nature's attempt at restoration of function in the face of permanent vascular lesions.

Pfeiffer (20) has shown similar conditions of disturbed vascularity of the brain in tuberculosis, where it is probably indicative of the mental changes commonly found in that condition. Old age of itself is not necessarily accompanied by much reduction in the complexity of the capillary regularity and arborizations, as will be evident from Fig. 3, from a case aged 90, which is given as an illustration of the normal for that reason. Some of the irregularities met with are described, with the method, in my paper in the *Journal of Anatomy* (16).

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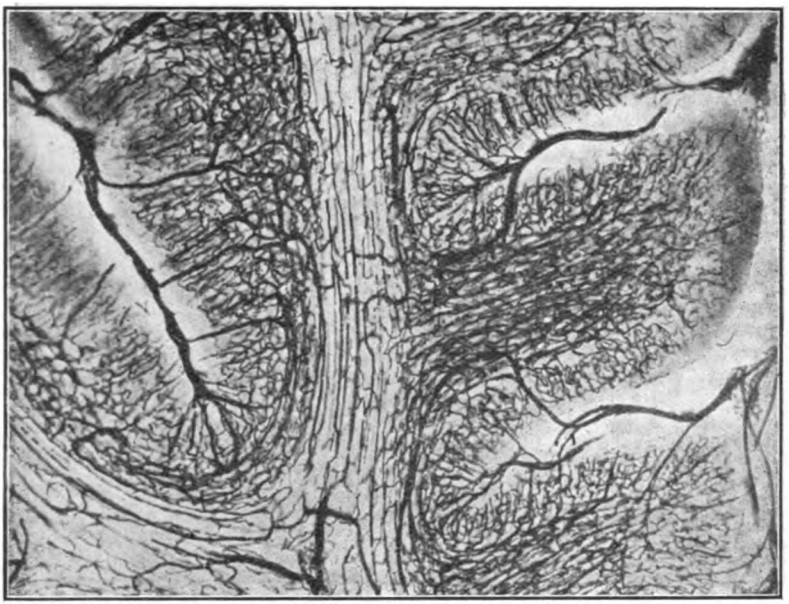

FIG. 2.—Cerebellum—selected area of normal appearance. × 24.

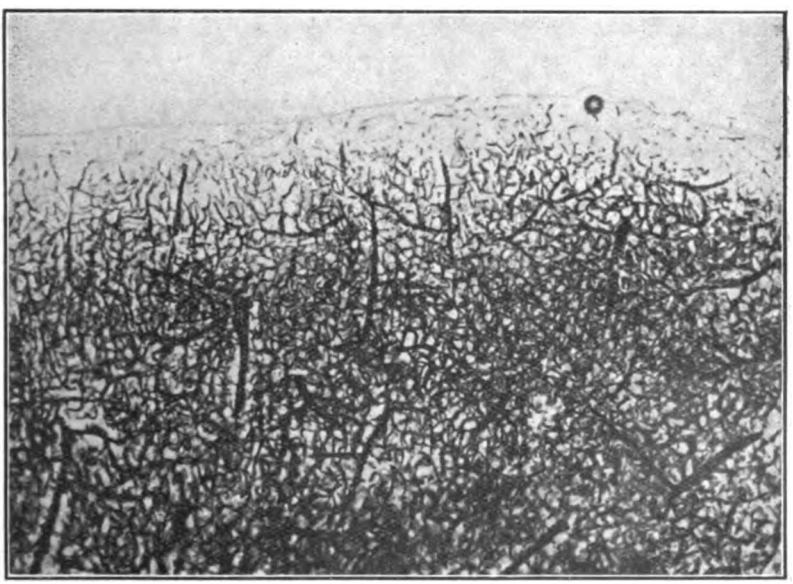

FIG. 3.—Cerebral cortex—selected area of normal appearance. × 25.

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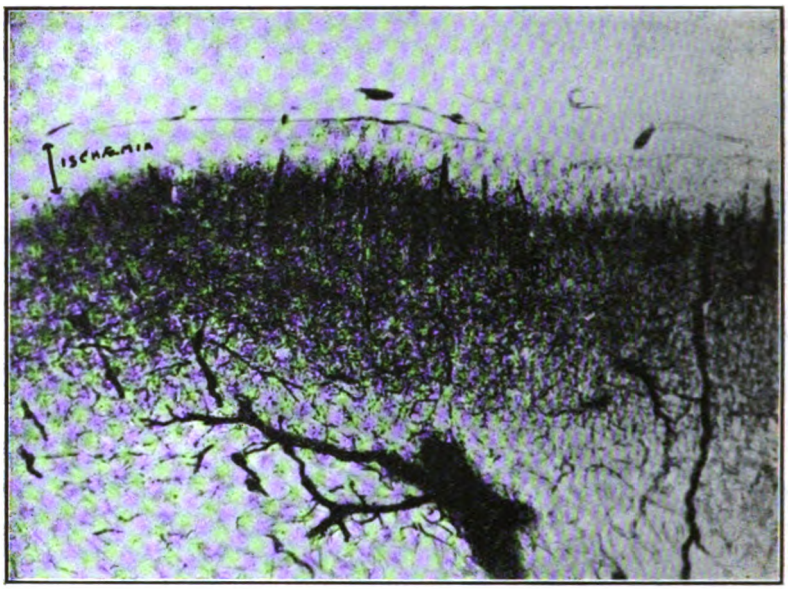

FIG. 4.—Ischæmia of outer cortical layer. (Case of general paralysis.) × 28.

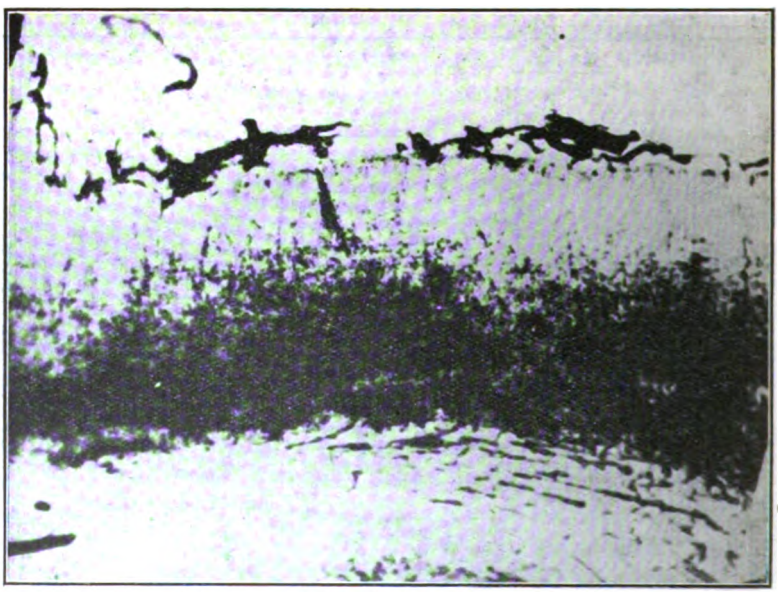

FIG. 5.—Ischæmia of outer cortex with congestion of deeper layers. (Case of melancholia.) × 25.

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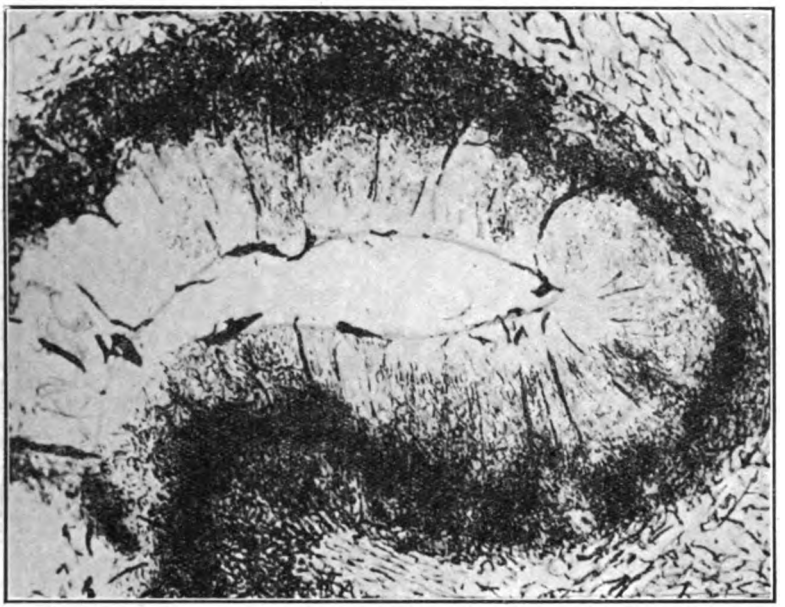

FIG. 6.—Ischæmia of outer layer within a small sulcus. (Case of melancholia.) × 10.

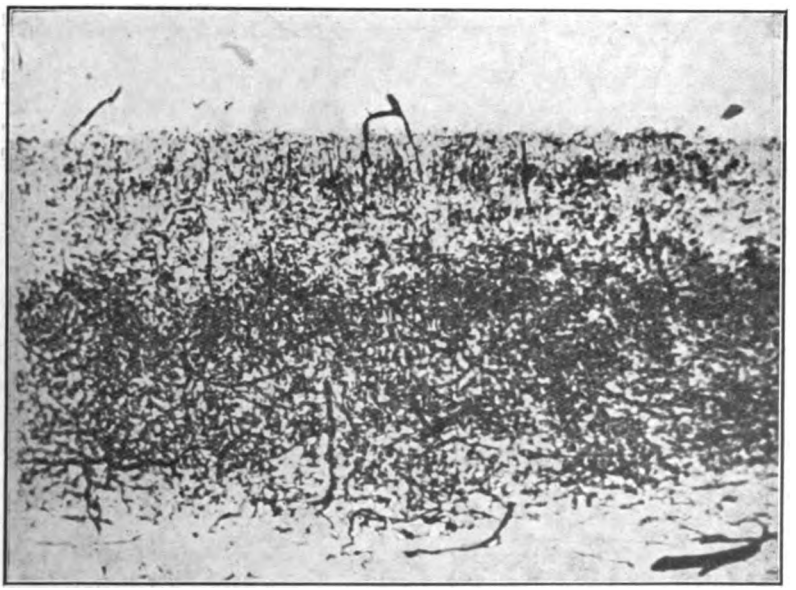

FIG. 7.—Cerebral cortex, congestion of outer layers. (Melancholia following post-lethargic encephalitis.) × 28.

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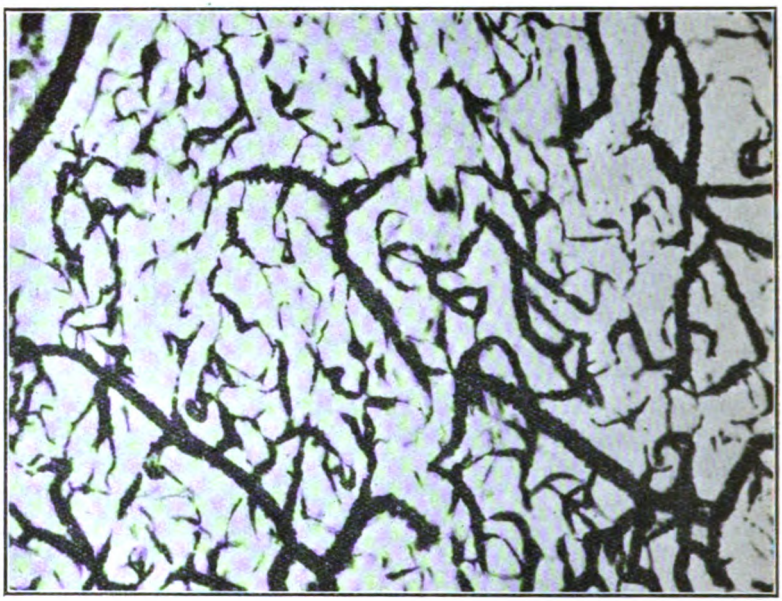

FIG. 8.—Diapedesis of corpuscles. (Case of epileptic insanity dying in *status epilepticus*.) × 40.

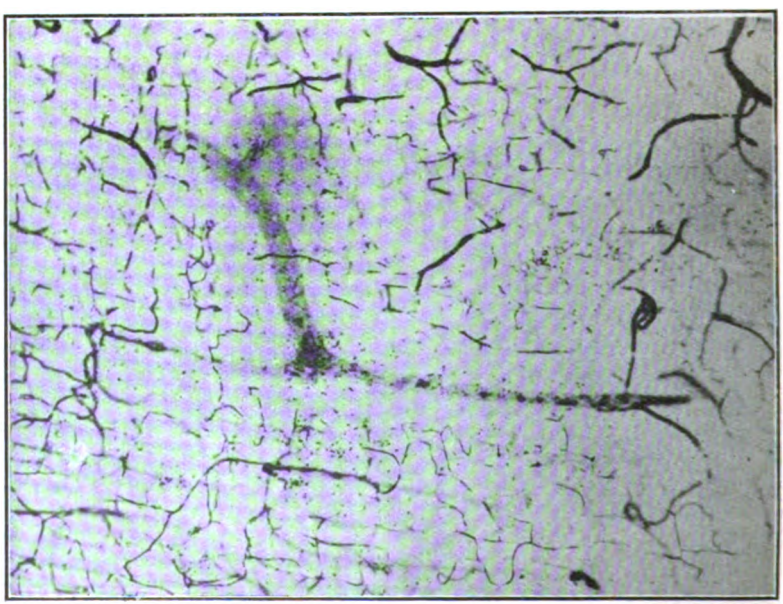

FIG. 9.—Thrombosis of capillary. (Case of confusional insanity with lobar pneumonia.) × 40.

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Fig. 4 shows ischæmia of the outer synaptic layer of the cortex ; it is from a case of general paralysis. Fig. 5 shows a more extensive ischæmia with congestion of the deeper layers of the cortex ; from a case of melancholia. Fig. 6, also from a case of melancholia, shows ischæmia within a small sulcus, and is given to show that these appearances are not due to mechanical interferences during preparation of the section. Fig. 7 shows congestion extending into the outer layer of the cortex and is virtually an encephalitis ; it is from a case of melancholia following an attack of encephalitis lethargica. Fig. 8 shows diapedesis of the cerebral capillaries in a case of epileptic insanity which died in *status epilepticus ;* he also had a lobar pneumonia. Fig. 9 shows thrombosis of some of the capillaries in a case of confusional insanity.

The table given shows that evidences of vascular disturbances can be obtained from all the brains examined in mental disorder cases. Further work is in progress, and it is hoped to correlate certain localizations of vascular defect with the major mental symptoms and with neurological conditions which the patients have shown during life. The theory which I have put forward of the limitation of synaptic function by the vascular state can be proved or disproved by a further application of the method, and besides opening up a more hopeful outlook in the barren field of treatment of certain neurological conditions, it requires that psychiatric cases should be treated as suffering from vascular instead of nerve pathology.

Correlations.

The use of focal ischæmia of the cerebral synapses as a common denominator of mental and medical science will now be illustrated.

Mental phenomena.—It has long been accepted that local ischæmia of nervous tissue means functional paralysis. Mott showed that temporary or permanent dementia could be produced in animals by carotid ligature. The Assyrians compressed the carotids of their young men to relieve the pain of castration. Trotter, with regard to cerebral hæmorrhage, says that irritative symptoms mean congestion and paralytic symptoms mean anæmia. Mental effort results in an increase in the oxygen and a decrease in the carbon dioxide of the jugular blood, i.e., a dilatation of cerebral capillaries. Gildea and Cobb described a loss of intelligence and a bizarre activity of cats whose vertebral artery had been ligated. Drugs producing volubility, exhilaration or motor hyper-activity (ephedrine and benzedrine) belong to the class of drugs specifically acting upon blood-vessels. Ferris denervated the carotid sinus, and some patients after the operation could see better and felt better, and colours appeared brighter. Royle cured irritability and bad temper by cervical sympathectomy—which effected a change in the cerebral circulation, and von Braunmühl showed excitement to be associated with capillary stasis.

Neurological.—Cerebral vascular spasm is said to be the cause of cerebral

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anæmia, eclampsia, migraine, functional hemiplegia, aphasia and amaurosis. Cortical ischæmia is considered to be the cause of the familiar Babinski sign (25) and the cerebral malformations in Pick's disease. Mott showed that firm pressure on the upper cord did not prevent nerve-impulses passing from the cortical leg area to the muscles, but light pressure in the lumbo-sacral region sufficient to cause ischæmia of the grey matter completely obstructed even the strongest impulses. The widespread functional loss in many nervous lesions (infantile paralysis) later recovers as the ischæmia caused by œdema is relieved. In addition to the relief of symptoms of mental dysfunction by cerebral stimulation, the associated contortions of torticollis, parkinsonism, etc., are also temporarily relieved; such clinical demonstrations show that not only mental disorder but many neurological conditions are caused by functional rather than structural disorder of the cerebral neurones, the synaptic pattern remaining normal under such diseased conditions, but a functional inhibition is imposed by deficiency in the blood-supply of the synapses. Stammering is illustrative of interruption of synapses by ischæmia where the neuronic patterns are obviously intact, and the locomotion and oculogyric crises of post-encephalitis show how activity of contiguous synapses may sometimes allow sufficient oxygenation of the ischæmic synapses to permit temporary direct function (also illustrative of the reason for the peculiar associated behaviour in both conditions). Failure to recognize the vascular regulation of synaptic function as being the basis of conditioned reflexes has led to difficulties in neurological research, such as the necessity for extraordinary precautions and unnatural isolation of animals during investigation.

Physiological.—The physiology of alternative reactions to the same stimulus is explicable by my theory of vascular regulation of synaptic function; for example, the same olfactory food stimulus can be a source either of attraction or repulsion, depending upon the full or empty state of the stomach, the visceral impulses presumably influencing the vascularity of alternate cerebral synaptic paths. In many animals (e.g., the early worm-fishes) olfactory nerves pass from the olfactory region both to the pineal-habenular and the pituitary-hypothalamic regions of the brain (Clark (22)). The reason for olfactory impulses passing to such widely different parts of the brain is difficult to explain by any other mechanism than the synaptic facilitation theory which I have put forward, and which makes the fact that a crab's nerve supplies both flexor and extensor muscles of the joint no longer a curiosity. Many of the discordant results encountered in the study of the action of certain nerve excitations, and of the pharmacological action of drugs, are possibly due to failure to recognize the all-importance of the vascular supply of certain synapses in order to get the desired pattern of reflex response. The lability of reflex pattern response is well shown in, for example, a dog, previously careful and obedient, who at the sight of another dog across the road at once becomes heedless of command and of traffic—the cerebral vascular capillary pattern

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having been "set" by the mental reflex to that which allows only the one nervous synaptic pattern and resultant behaviour.

Emotional.—The emotions of shame and embarrassment are associated with peripheral dilatation, and fear, pain and anger with peripheral vaso-constriction—no doubt a corresponding alteration of certain focal brain areas occurs simultaneously. Stimulation of the anterior hypothalamus in the region commonly known as the fear area contracts pial arteries (Stavraky (23)), and pain inhibits function and peristalsis. Obliterative endarteritis is distressingly painful, and thus cerebral vascular spasm may be evidenced by melancholia. Continued pain may be compensated by vascular adjustments similar to the extinction of the sound of clock-ticking; in such conditions the collateral paths of the pain-impulse which cause vascular spasm may persist—in chronic nasal sepsis a facial pallor is commonly noticeable. Similarly mental effort and metabolism may be inhibited by chronic pain stimuli, where the actual sensation of pain has, by reason of its duration, ceased to enter consciousness.

Endocrines.—That endocrine secretions affect the vascularity of the brain has long been known, and Cushing's (21) demonstration of the intra-ventricular action of pituitrin is very convincing. Schmidt (17) has studied the elective action of certain endocrine secretions (pituitrin, adrenaline) upon different parts of the cerebrum and brain-stem. Although endocrine activity has no direct relation to the insane condition (as evidenced by cases of extreme endocrine hyper- and hypo-activity without mental disturbance), yet its effect upon a case with a disintegration of the capillary pattern is of the utmost importance. This is best illustrated by contrasting the maniacal behaviour in some cases of hyperthyroidism (in which the endocrine secretion is acting upon an already disintegrated cerebral capillary pattern) with the sexual behaviour of an animal having a normal brain, but with total removal of gonads and genitalia, on injection of the sex hormone.

Stupor.—In stupor and hibernation, function and production of metabolites are minimal and the synapses are accustomed to less oxygen requirement. A blood-supply adequate to support existence in hibernation would probably result in anoxæmic necrosis of tissue in the normal state of function. Stupor and apathy are means of forced economy of the cerebral blood-supply under conditions where it is limited by spasm or disease.

Mental deficiency.—The anthropologist Hrdlicka (24) said that man had not yet progressed to his full mental capacity because the development of the cerebral vessels had not kept pace with the development of the brain. It follows from this that any additional lack of development of cerebral vessels must necessarily cause a degree of mental defect depending upon its locality and extent. Early traumatic or toxic disturbances of the fœtal cerebral circulation inhibit growth of embryonic cells which, owing to their period restriction (Needham), cannot later develop even should the circulation be

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later restored. Evidences of such are seen as "stigmata of degeneration", and a restricted locality is indicated by "idiots savants". After birth the conditioning of environment influences the cerebral vascular supply by the constant buffetings of emotional and digestive upsets, and to these may be added dietetic and infective causes of vascular disease, also the later vascular diversions of puberty, menstruation and courtship. Vascular spasm persisting during development (such as from otitis, sinusitis, naso-pharyngeal sepsis, etc.) necessarily inhibits mental development, and such cases, even although passing as normal, would have been superior mentally and physically had such accidents not occurred.

Psychiatric.—The individual personality is built up by neuronic patterns which are conditioned by social demands, and it follows that when such pattern is disrupted by chronic ischæmia, the behaviour will change away from such properly conditioned types to inferior or antagonistic (asocial or negativistic) reactions. The naughtiness of post-encephalitis is a typical example. In my work I frequently find atheromatous changes in the vertebral and basilar arteries, sufficient to form a prestatic condition of the lower vital centres; ischæmia of these centres would occur by dilatation of branches of the posterior cerebral artery, which, as Williams showed, has its sympathetic nerve-supply from the carotid and not from the vertebral. Since the dilatation in such conditions must be overcome by a vascular agent of constriction, stimulation of the fear area has a clinical significance.

In addition to the pale yellow areas in the basal ganglia and cortex, which I have noted are universally present in mentally disordered cases and which afford naked-eye evidences of ischæmia, the following associations are of interest: *Epileptic insanity* may be regarded as invariably associated with cerebral vaso-lability, and parenchymatous degeneration in certain brain areas (cornu ammonis) has long been recognized by Spielmeyer and others, as due to cerebral vascular spasm. Mott obtained epileptic phenomena by moderate stimuli of the motor cortex in animals also suffering from experimental cerebral ischæmia. Kausmaul and Tenner compressed the carotids of six persons, two having epileptic fits. Hughlings Jackson thought epilepsy was due to vascular plugging of small cerebral vessels. Kennedy noted blanching of cerebral capillaries during epileptic tonic spasm with engorgement in the clonic phase. Penfield says the constant visible phenomenon in the brains during a seizure is the cessation of arterial pulsation in a focal area. *Schizophrenia* shows peripheral capillary phenomena comparable with ergot or histamine poisoning as pointed out by Bayard Holmes; Hollander and Rouvroy described thickening or atrophy of walls of minute vessels. Gottlieb ascribed the static vascularity present to an irresponsiveness of the sympathetic system. *Manic-depressive* insanity shows coagulative necrosis of nerve-cells (Marburg)—probably due to ischæmia; Bouman and Brouwer found dot-like hæmorrhages in the cortex, hyperæmia of the pia, and thrombi

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partly extending from the pial veins to the large cerebral sinuses in two cases of acute delirium. In *confusional insanity* Damaye and Poirier described a meningo-encephalitis similar but of less intensity to that of general paralysis. The vascular lesions of *general paralysis* are exceptionally severe, as shown by my method. Winkelman and Eckel remark on the similarity of the vascular lesions of syphilis and sepsis. As the pathological causes of ischæmia increase, neighbouring ganglion cells may become destroyed, resulting in dementia or permanent neurological lesion. *Dementia* is of two types: The one due to ischæmia of synapses and clinically distinguished by an ability to show temporary recovery or cerebral stimulation; the other due to ganglion cell necrosis, distinguished histologically by atrophy and thinning of widespread cortical areas.

The success of the various treatments of general paralysis in most cases depends upon their ability to alter the vascularity of certain brain areas. My work with the capillary stain method has led me to suppose that mental disorder syndromes are due to pathological obliteration or disruption of the vascular capillary pattern upon which the nervous synaptic pattern depends for normal function. A bizarre conduct ensues when function is compromised by unusual nerve routes, and secondary degenerative changes, which occur when the ischæmia is widespread, result in acidic and excitatory changes, causing irregular stimulations of the sensorium or impulsive behaviour. The various psychiatric syndromes can be assumed to depend upon the anatomical localizations, the multiplicity of areas involved, the age of onset, the duration and the oscillatory vascular changes consequent upon attempts at overcoming any ischæmia of vital areas, which must at all costs function to preserve life itself.

Biochemical.—Local cerebral ischæmia causes œdema, which shuts off anastomoses, and therefore the brain suffers both oxygen lack and accumulation of metabolites; one of these, adenylic acid, has been shown by Drury to cause capillary dilatation. Under conditions of ischæmia lactic acid is produced, which physiologically reduces the oxidation-reduction potential of the nervous tissue, with protective cessation of function; it is probably for this reason that there are naturally alkali (ammonia)-producing enzymes in the brain. Under pathological conditions of ischæmia the acidity may exert a poisonous effect upon neighbouring tissues, causing fatty changes in the cells, as found by Cotton, or mucin accumulations (Buscaino); mucin has been found by us in the brain of a case of myxœdema. Anoxæmia also causes loss of potassium ions, which have a powerful effect in inhibiting adjacent nerve-function, and the finding of an increase of potassium in the cerebro-spinal fluid in 8 out of 20 cases of schizophrenia by Katzenelbogen (26) thus indicates cerebral ischæmia.

Bacteriological.—Repeated bacteriæmias of chronic sepsis, such as established by Parish and Okell and Round and Kirkpatrick, are related to ischæmia and vascular clogging by well-known bacterial reactive mechanisms, such as adhesion of organisms to the cerebral capillary wall (found by me in a

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case of acutely excited dementia præcox), which, but for the rapid disappearance of organisms (about four days), would be much more frequently found; the tissue fixation of organisms (Menkin); the inflammation caused by antibodies and antigen uniting in the tissues (Opie); the progressively increasing local reaction to repeated doses of the same antigen (Arthus); the clogging of vessels with antigenic material (Kahn); and the peculiar reaction to 24-hour spaced injections of bacterial filtrates (Schwartzmann). A chance involvement of a certain local tissue renders it liable to become a *locus minoris resistentiæ* in the event of similar bacteriæmias on a future occasion. The same occurs from contiguous diffusion of neighbouring infection. A new field is opened up for the action of toxins upon the vascular supply of special focal areas of the brain.

Atherosclerosis.—Arteritis may be caused by typhoid fever, diphtheria, measles, scarlet fever and small pox. My researches on the Widal reaction show that a significantly high proportion of mental patients (including defectives) have had infection with dysenteric or food-poisoning organisms. Atheroma of the cerebral arteries is commonly found, often with partial thrombosis of the larger arteries; in one case a complete thrombosis of the carotid artery from the junction with the external carotid to its intra-cranial termination was found. Lead poisoning causes arterio-sclerotic changes by reason of the vascular spasm; it is probable that other causes of spasm, such as the referred pain of chronic sepsis of the ear, nose and throat may similarly be related ætiologically to the atherosclerosis commonly found. Many specimens of nasal sinusitis also show athero-sclerotic changes of the internal carotid artery, and are fully described in my book.

Nasal sinus sepsis.—At Birmingham the incidence of nasal sinusitis and its association with mental disorder has been investigated and a valuable collection of museum specimens made. The lymphatic drainage of the nasal sinuses passes into the deep cervical chain in relation to the superior cervical and the carotid artery; contiguous toxins diffusing into the ganglion or perivascular sheaths affect the cerebral circulation by spasm, stasis and thrombosis in a manner analogous to that shown experimentally by Orr and Rows. Pituitary gland swelling due to contiguous sphenoidal sinus sepsis may press upon the tuber, thus causing a reflex dilatation of the pial vessels.

Focal cerebral ischæmia thus forms a useful connecting link between mental and medical scientific research, and opens up new possibilities in the prevention and treatment of mental disorder and defect.

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Discussion.

Prof. F. L. GOLLA said he was sure all must admire the courage and industry of Dr. Pickworth, perhaps especially his courage. That gentleman had done what very few members dared to do. All, no doubt, kept some sort of note-book of things which could be philosophized about, but there the matter usually stayed. It was a pity it should so stay, and many of the points which Dr. Pickworth had brought out and which could be thought about were those with regard to which Dr. Pickworth might be right, and he might be wrong. As a technician Dr. Pickworth had shown superlative skill. Many of the demonstrations given by him were not to be regarded as final; they might be taken as suggestions of what might happen if certain views were adopted. Understood in that spirit, he was sure all present would have learned much from his most interesting lecture and demonstrations.

Dr. J. H. MACDONALD said that what had been given to the meeting to-day had been extremely interesting, and perhaps it had been informative too. It was a very old question, and was one of those which Ford Robertson, senior, and himself were interested in when they were doing histological work on the degeneration of the nerve-cell. It was striking to him, the speaker, as a pathologist, how often he came across vascular disturbances in cases of neuronic disease. Were these changes in the cell primary, or were they secondary? Could he be told whether these toxæmias were acting primarily on the nerve-cells, or on the capillary vessels? Should they be regarded as vascular changes, or as neuronic? That was, he thought, still an unsettled question. In many cases, however, one was able to find evidence of circulatory changes, and workers were beginning to lay great stress on the presence of ischæmia.

Prof. Golla had uttered a very important warning when he spoke of the defective methods of estimating the circulation in a particular cerebral area. It was very important to reflect on the ischæmic changes which followed concussion and also emotional shock. Many suffered periods of concussion, others suffered enormous emotional upsets. Because they had such experiences he would not consider it showed they were on the way to becoming lunatics, via these ischæmic changes.

Another point which struck him as important was the reference to epilepsy and the changes in the cornu ammonis, which could be interpreted as ischæmic. He asked what was the interpretation of the epileptiform convulsions in general paralysis, where the changes were regarded as congestive rather than ischæmic? Gross congestion seen might be accompanied by local ischæmia; but the picture of an epileptiform convulsion was not one of ischæmia. There existed a striking want of co-ordination between these pathological and histological findings in reference to the clinical conditions. What was seen could easily be explained wrongly. The clinical and pathological pictures did not fit in with each other sufficiently to warrant a general conclusion yet. As Prof. Golla had just said, it was necessary to be very careful about the conclusions based upon these findings, otherwise one might very well be led astray.

Dr. J. H. BIGGART expressed his congratulations to Dr. Pickworth on the

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amount of work he had done. Four years ago he would have been much more interested in it. He had been examining 300 brains a year, and while at first he was inclined to explain dementia on pathological findings and on the vascular disease which he found, he soon came to the conclusion that he was finding in non-demented patients vascular pictures equivalent to those in the demented: atheroma, arteries sclerosed, and areas of ischæmia, with infarcts; in fact all the vascular lesions which Dr. Pickworth showed to-day could be found in routine examinations of brains of patients dying in a general hospital who did not suffer from dementia.

He had been very interested in the application of Dr. Pickworth's stain, but the view of that gentleman was anatomical more than pathological. There were many artefacts in reference to the pathogenic aspect of mental disease. There was the handling of the brain itself, and there were the agonal changes. He had a patient in hospital who died from asphyxia due to a large retro-sternal goitre. Her brain showed many vascular pictures resembling those which Dr. Pickworth showed, but because she had sudden terminal asphyxia no evidence was forthcoming in regard to those vascular changes and the possible cause of her dementia præcox. Therefore it was necessary to view with great caution any explanation of mental disease which was based upon these pictures. Other explanations brought forward included swelling of the sphenoid. He had found that often in ordinary mental cases, but not causing any symptoms which could be associated with any damage to the tuber. It was known that damage to the tuber produced gonadal atrophy and diabetes insipidus, and these cases of sphenoidal sinusitis with pituitary œdema did not come into this category. He challenged the reader as to whether there was tuberal damage, causing damage to the tuberal vessels.

His experience in examining 400 control brains made him sceptical about the conclusions which might be based upon implication of the sinus. He felt there might be an association. He owed much to Dr. Pickworth, who had been very brave, but he, the speaker, did not feel positive about it. He felt himself to be in a position of negativism with regard to a possible explanation.

The PRESIDENT said that Spielmeyer had retracted all he had previously said about the effects of functional disorder of the vascular system. A good deal was taught about toxæmia in mental disease. He always said that it depended, first of all, on the amount of the toxin, and, secondly, on the constitution of the individual. If twelve men were placed in a row and half a glass of whisky was given to each, not much difference might be noted in the effect it appeared to have. If, however, the quantity was increased to two or three glasses, one noted that some of the men got confused, then garrulous; one might become arrogant and wish to fight; another became unduly affable and familiar. Later the alcohol caused sleep. Clinically one could produce different effects according to the quantity taken, and eventually very small amounts might be injurious. Because a great variety of clinical conditions could be demonstrated it must not be said that the lesions were connected with mind; they were connected with motor and sensory areas which controlled functions in other parts of the body, such as the endocrine glands. He did not see how any kind of injury could produce a specific mental symptom.

Dr. PICKWORTH, in reply, dealt first with Dr. Macdonald's remarks on agonal changes. He, Dr. Pickworth, had compared the results in cats which had been killed in various ways, such as by strangulation, decapitation, cyanide or gassing, and he thought the agonal changes soon disappeared, and that the ischæmic areas which he had demonstrated were chronic changes.

With regard to the question whether a toxin acted on the vessels or on the cells, if the toxin acted on the cell the latter was unlikely to recover, and if it did one would not expect it to revert later to the identical toxin-impaired state in the same way as the mental state reverts after temporary restoration of sanity by methods of cerebral stimulation. If one gave an excess of CO_2 a patient would

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become mentally normal for a short period, but shortly after the effects of the stimulus passed off the original state was resumed. It could therefore not be an effect on the nerve-cells as the functional capacity of nerve patterns in mental disorder remained normal.

Another point was that ischæmia was not necessarily a permanent change. There were exhibited slides illustrating regeneration of vessels. One had always been taught that nerve tissue could not regenerate, though vessels could do so; ischæmia could be a temporary change. A physiological conditioning of the mental state or emotion by a definite capillary mosaic allowed of a definite neuronic pattern by inhibiting unwanted synaptic paths. There were millions of nerve-cells, with countless millions of synapses, but as long as only one capillary pattern existed, motor response to stimuli could only be along the one path left open for it.

With regard to general paralysis, he had never found a case of it without marked cerebral ischæmia, though congestion occurred at the same time.

Almost half his paper was devoted to the correlation of the clinical with the pathological for which speakers had asked.

Referring to Dr. Biggart's remarks, dementia also could be associated with ischæmia, but was of two types, one recoverable, the other permanent with destruction of nerve-cells. Spielmeyer and others had shown that there could be ischæmic necrosis of tissue, and there must be a consequent dementia, because the nerve-cells had gone. That could be seen best in cases of G.P.I., where the atrophy of the brain was evident to the naked eye.

With regard to changes in the tuber, it was not his intention to infer there were any permanent changes there. Any contiguous sphenoidal sinusitis might cause inflammatory changes affecting the local blood-supply, and a very small change might produce sufficient ischæmia to cause functional inhibitions. It could be shown that light pressure on the tuber caused dilatation of the pial vessels.

Referring to the President's remarks, he, Dr. Pickworth, placed the whole theory of mental and emotional states on the capillary patterns. An individual personality resulted from labile changes in capillary pattern, conforming to circumstances and allowing appropriate behaviour. The synapses might normally become adjusted to one pattern, but that pattern changed conformably to emotional reaction, mental effort or to any instinct, or any hormone which affected the vascular capillary pattern of the brain. Mental changes were not necessarily associated with insanity, even if they were due to pathological ischæmia; and pathological changes could arise from undue prolongation of physiological causes. No doubt even alcohol acted primarily on the cerebral capillary circulation, because one of its effects was to cause inconvenient changes in the capillarity of the face.

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THE CIRCULATORY FACTOR IN THE PATHOGENESIS OF MENTAL DISORDER.*

By E. KRAPF, M.D.Leipzig & Buenos Aires, F.R.F.P.S.Glasg.

THE circulatory factor in the pathogenesis of mental disorder can be dealt with in at least two different ways. The exposition can be narrowed down to a mere description of what we know for certain about the psychiatric significance of circulatory disturbances. On the other hand, I think it not less legitimate to look at the question as a highly fascinating problem, the full bearing of which upon psychiatry we are only just beginning to discern as a dim shape on the scientific horizon.

I should like to take the second of these two ways of approach. My object in taking this decision is certainly not the futile one of trying to erect pleasant, if lofty castles in the air; I am aiming at the much more practical goal of showing where our present knowledge seems likely to lead to further progress; and therefore it is perhaps not only justified, but even necessary to start with a short survey of the available basis of facts.

If I offer a short survey, clearly it can only be a very superficial one. The time is definitely past when a paper on circulation and mind could be limited to a few considerations on the possible ætiological significance of valvular disease or so-called arterio-sclerosis for certain not very characteristic confusional states. Since we have started to free ourselves from the hypnotizing influence of organological thinking, and now try to replace static ætiological by dynamic pathogenetic interpretations, we have evidently reached a much deeper insight into the real significance of the circulatory factor. Indeed there is hardly a chapter in mental medicine to-day where its interference could be wholly denied offhand, and many are the nervous and psychic disorders which seem to be definitely subjected to its powerful pathogenetic influence.

To refer only to the latter now, it is significant that the most instructive specimens of mental disorder depending on circulatory disturbances are not to be found among patients with coarse anatomical findings (such as arterio-sclerosis or syphilitic arteritis). On the contrary it seems that chronic degenerative or inflammatory alterations of the brain-vessels lead only to a rather unspecific chronic deterioration of the personality with a wholly

* Read at the Annual Meeting of the Royal Medico-Psychological Association at Ayr, July 8, 1937.

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uncharacteristic general dementia as the terminal state. On the other hand, there are many indications to show the remarkable pathogenetic significance of so-called functional circulatory disorders, i.e., of more or less sudden alterations of the blood-flow, which would of course affect the nutrition of the functioning nervous parenchyma.

There is no reason why a preferential role should be attributed to any special kind of functional disorder, e.g., angiospasm. Physiologists and pathologists have discussed the point intensively and are still very far from agreeing on it. In my opinion the clinician is not bound to pay too much attention to such theoretical controversies, as long as the main issue, the existence of dysfunctions of the brain vessels, is not seriously doubted. This is certainly not the case. Whatever the main disturbance, whether angiospasm, stasis, " peristatic " state or anything else, it is undeniable that the function of the brain vessels can be affected by mechanical, chemical and nervous stimuli, and that the vasomotor disturbances, thus originated, are able to impair the normal activity of the brain.

We owe this certainty mostly to the brilliant work of two eminent pathologists, Gustav Ricker and Walther Spielmeyer. Ricker, the founder of the so-called " Relationspathologie ", showed the significance of vasomotor troubles in general pathology. He demonstrated the nature of vascular reactions to different stimuli, insisted on the importance of persistent irritative states in the nervous supply of the vessels, and studied the detrimental effects of vasomotor disturbances on the function-subserving parenchyma. With the brain he dealt only occasionally, but showed nevertheless that the cerebral vessels seem to obey similar laws to those obeyed by other circulatory territories. Spielmeyer and his collaborators occupied themselves exclusively with the brain. Their principal merits consist in having found histological appearances indicative of functional circulatory disorder, and in the way they patiently followed up these " signals " in pathological processes of very different kinds and descriptions.

It need not be emphasized that Spielmeyer's work (an account of which was given by the author himself at the Oxford Meeting of the Royal Medico-Psychological Association in 1930) is of more intimate concern to the clinical psychiatrist than Ricker's. In fact there is hardly any finding in modern brain pathology which could be compared in its direct clinical significance with Spielmeyer's main achievement, viz., the histological demonstration of pathogenetic identity in so varied a series of morbid conditions as head injuries, various intoxications and infections, hypertension, epilepsy, certain excitement states, etc., etc.

Pathogenetic identity is, of course, not complete identity. It is utterly impossible to compress Spielmeyer's concept into a blunt statement that it is " all more or less the same " or any other platitude of the kind. We may be satisfied, however, that pathogenetic identity is important enough. Even if

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the first links in the causal chains seem to be different with different ætiological factors, there is still a sufficient amount of histological evidence to prove that eventually they all meet in the final common path of functional circulatory disorder, i.e., that they are all identical as regards the last link, the ultimate cause of actual disturbances in the nervous substance itself.

Let us stop here a moment and ask the first question that has a direct bearing on clinical problems. If the same " last link " is really at work in so many nosologically different morbid conditions, is it not to be expected that a certain resemblance would also appear in the corresponding clinical pictures ? It seems highly probable that this is the case. Thiele (in his paper on circulatory troubles and psychoses) says very rightly : " When various exogenous noxæ meet in a common series of events which may be considered—at least pathogenetically—as an intermediate link very close to the cerebral processes themselves, then it seems comprehensible that the corresponding clinical pictures should appear to be intrinsically related to each other." Nevertheless all this is only theoretical argument. The real question is, is there any clinical evidence which bears out our hypothetical assumptions ? This is in fact the first point we must try to ascertain.

In dealing with this fundamental problem I may base my exposition, at least to a certain extent, on some personal research. Several years ago I started an investigation into the still largely unexplored field of the psychoses associated with arterial hypertension. I tried to find out whether such a thing as a hypertensive psychosis really existed, and, in the event of this being the case, what would be its relation to other kinds of mental disorder, especially to arteriosclerotic disturbances.

A survey of several thousand cases brought me to the conclusion that arterial hypertension actually has psychiatric characteristics of its own, and that the clinical picture in hyperpiesis is very different from that observed in " pure " (non-hypertensive) arterio-sclerosis of the brain vessels. An explanation for these results was furnished by a more thorough investigation, including the use of vascular tests (such as F. Lange's " Nachströmungsversuch "). It could indeed be shown that the actual cause of mental trouble in my hypertensive patients was not to be found in a more or less marked elevation of their blood-pressure, but in that definite exaggeration of vascular lability which is so typical, especially for the earlier stages of hyperpiesis, while " pure " arterio-sclerosis is characterized by a more or less complete absence of even the normal amount of vascular response.

I am inclined to believe that these special results arrived at in hypertension have a certain value for the solution of the more general problems that are interesting us here. The pathology of hyperpiesis being what it is, the study of the psychoses associated with this condition seems to offer a very useful instrument for going into the whole range of psychic disturbances of vasomotor origin.

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I cannot go into the details of clinical description here.* The points which are of general interest can be expressed in two very condensed statements:

(*a*) The hypertensive disturbances show a marked tendency to make their appearance in "attacks", i.e., in more or less short episodes which often lead to full recovery.

(*b*) They seem to prefer certain psychopathological syndromes, such as—

(1) short losses of consciousness of the *petit mal* type often observed in epileptics (see also my previous paper on "Epilepsia tarda");

(2) relatively short twilight states of varied clinical appearance, but always characterized by a high degree of confusion;

(3) transitory states of anxiety and depression, ranging from the ordinary "bad mood" up to the deep depression of the "vital", endogenous type.

The next step, I think, is perfectly obvious. If all mental disturbances due to circulatory disorder are in fact clinically alike, it is to be expected that the principal patterns of hypertensive psychoses will also be found in other psychoses of circulatory pathogenesis. In other words, hypertensive mental disorder must be something like a "model" for circulatory mental disease. Is there evidence that this expectation is confirmed by clinical facts?

The evidence we get is quite conclusive indeed. Of course I cannot go into all the details here. It would easily fill a book if I began to compare in a really painstaking way the clinical pictures of, say, epilepsy and migraine, carbon monoxide and lead poisoning, pellagra and head injury—not to speak of rarer events such as the psychoses which sometimes accompany the so-called "vasomotor neuroses" (like Raynaud's disease, etc.).

On the other hand, I have not yet investigated all conditions of supposedly circulatory origin on a sufficiently large scale. Nevertheless I may say that in all instances where a conclusion seemed admissible, course and clinical picture clearly showed the "circulatory" features I have described.†

Considering how unorthodox the foregoing statements are, I would not blame anybody for reserving his judgment or for asking sceptically what was the practical value of this argument. It is certain that the clinical resemblances go a long way. In some instances they even go beyond the purely clinical field, and enter the territory nowadays called phenomenological. Among the more typical psychopathological features of circulatory mental disorder I might mention a special kind of cœnæsthetic disturbance which often occurs in depressive states, and the very marked inclination of many patients to "see red" when emotionally excited, or to hallucinate fire and fiery red objects when passing through a confusional episode.

* *Vide* Mayer-Gross's paper in this issue of the Journal; also Guttmann's paper on "Hypertension" (1936).

† *Vide* Guttmann's paper in this issue of the Journal, which shows that clinical observation in Raynaud's disease, migraine and head injury well fit in with my conceptions.

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On the other hand, I am quite willing to accept the objection that the resemblances just mentioned are of a somewhat unusual nature. Looking again at the circulatory syndrome triad in question, we notice promptly that only the *petit mal* and depressive states seem to be more or less clear-cut syndromes, whereas the clinical pictures I referred to as twilight states constitute in fact another rather varied group of syndromes, comprising not only soporose and delirious pictures, but frequently also epileptiform, catatoniform and even hysteriform types of confusion. If we consider now that we started with the assumption that a single pathogenetic agent is responsible for all these different-looking disturbances, then we realize fully that there are some very serious questions still to be faced.

I do not think there are many ways of looking at our problem. If the pathogenetic stimulus is supposed to be of the same character in all cases, the differences of appearance can obviously only be due to one of the following four variants :

(*a*) The stimulus may be more or less intense.
(*b*) The brain may be more or less susceptible.
(*c*) There may be localized disturbances in certain vascular regions.
(*d*) There may be a localized vulnerability in certain cerebral regions.*

I shall try to show that probably each of these factors plays some part in the pathogenesis of circulation-conditioned mental disorder, and that the confusing variability of clinical appearances observed may quite satisfactorily be explained by the assumption of a joint and concurrent activity of various factors at the same time.

Regarding the " intensity factor ", I may limit myself to a few words. It is indeed quite noteworthy that, when Specht first insisted on its significance, he did so actually in describing the mental effects of carbon monoxide poisoning. Specht's formula, " high intensity—confusional states ; low intensity—depressive states ", has since then often been confirmed, and it is immediately evident that the circulatory factor has a special tendency to act in the manner mentioned.

Concerning the general susceptibility of the brain, I should like to be a little more explicit. The basic fact in this respect is probably Reichardt's finding that some individuals are liable to develop a sort of swelling of the brain (not identical with œdema, and called " *Hirnschwellung* " by the author). According to Liesegang this swelling may be interpreted as the swelling of a hydrophil colloid treated with a very dilute acid. As nutritive disturbances of the nervous substance actually produce slight acidification in the tissue, we

* It is obvious that this formulation does not cover more than the purely patho-physiological aspect of the problem. There is another source of symptomatological differences emerging from the internal life-history of the individual. Especially the so-called " content of the psychosis " depends mainly on the previous experience of the patient—or to put it otherwise, on his biography and his self-interpretation. It is impossible to deal fully with this psychological aspect here.

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may safely assume that circulatory troubles will be among the most important stimuli to provoke *Hirnschwellung*. The clinical facts fully confirm this hypothesis. Wherever we have reason to suspect a circulatory pathogenesis in a disease we find candidates for *Hirnschwellung* among the patients. This is obviously a very important point, because the capacity of the brain to react with swelling can now be interpreted as a function of its physico-chemical constitution. If we consider that according to Reichardt there is a close connection between *Hirnschwellung* and mental disorder of epileptiform and catatoniform appearance, the immediate clinical significance of his pathological findings is clear.

The local factors are probably even more interesting than the general ones. In saying this I am not even referring to the localization problem itself. I think that the study of character changes following frontal lobe lesions, the ample material furnished by the encephalitis epidemics of the last two decades and such single findings as Lhermitte's " peduncular hallucinosis " have definitely shown that mental symptoms and syndromes may have a localising value. But there is also a pathological and pathogenetic problem to be solved; in other words, we must ask ourselves whether the vascular supply or the physico-chemical organization of a given focus should be considered as primarily responsible for its isolated affection.

It may well be that further investigators will come to the conclusion that Spielmeyer's " vascular type " and Vogt's " pathoclisis " are not so completely incompatible in reality as they seem to be in theory. A. Meyer's recent studies on the selective vulnerability of the globus pallidus have shown that it is not impossible to reconcile the two opinions. The troubles we call circulatory now are apparently mostly due to the oxygen deficiency brought about by the (possibly temporary and incomplete) interruption of the blood-flow. On the other hand, it is clear that such a deficiency is much more likely to arise in a focus which on account of its particular physico-chemical organization has an especially high oxygen need. Meyer is probably right in assuming that vascular and physico-chemical factors are far from mutually exclusive, and that both may be at work at the same time.

There can be little doubt of the great significance of this point of view. Generally speaking we reach a new aspect of our problem by enlarging the field of disturbances to be taken into account. It is not impossible that the " circulatory factor " of to-day will only be a part of the " oxygen deficiency factor " of to-morrow.* Even the localization problem itself is influenced by such ideas; for it is easily conceivable that the special physico-chemical organization of a given focus may be a hereditary pathogenetic factor.

In this connection a significant observation was published some years ago by J. Lange. This author had the opportunity to examine a family where

* *Vide* A. Meyer's paper, summarized in this issue of the Journal.

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the psychoses observed (including even a case of general paresis) invariably took the form of a catatonic syndrome. In two members of this family a microscopic examination of the brain was made ; and in both these cases there were slight histological alterations in the putamen. Personally I think that the systemic vulnerability was not the only hereditary factor in this family. I assume that an inherited vaso-lability played the part of what I have called an extracerebral *Anlage* for mental disorder.*

It is interesting, though, that one member of the family (Emilie K—) seems to have had an arterial hypertension, and that her psychosis may well be interpreted as the effect of localized circulatory disorder of high intensity in a systematically vulnerable brain.

To summarize, it is certainly evident that one and the same pathogenetic agent is able to express itself in the most varied ways, and that the whole range of clinical pictures comprised in the triad of circulatory syndromes can be brought about by functional circulatory disturbances. But is this really a conclusion ? Is it not rather the starting-point for fresh problems of an even more complicated nature ? If we look at our material in the light of the above considerations, every observation becomes in fact a physiological problem by itself ; and this applies not only to cases of undoubtedly circulatory origin, but also to practically every other patient suffering from an episodic disturbance of " circulatory " mental symptomatology.

I do not deny that at first sight this generalization seems somewhat daring. Can we really compare a " constitutional ", " endogenous " disorder like manic-depressive insanity with an " organic ", " encephalopathic " disturbance such as, say, pellagra ?

I think it is an advantage of the method of thinking followed in this paper that it allows and even forces us to do so. The study of the circulatory factor induces us to approach our problems in what might be called a strictly causal manner. In a causal psychiatry there is no room for comfortable and vague adjectives like " constitutional " or " endogenous " or others of this type. The only question that matters is in fact that classical one of Rieger's : " What happens in the brain " ? It is clear that we are equally justified in putting it whether we deal with a manic-depressive or a pellagrous or any other kind of patient.

It is very tempting indeed to proceed along the lines I have tried to sketch. To say only a few words on manic-depressive disease, there are many interesting facts already known. I might remind you of Rosenfeld's studies on " vaso-motor psychoses ", of Dattner's ideas on the pathogenesis of anxiety, of the vasomotor lability at the beginning and the end of depressions found by

* I believe that in analysing the constitution of a mental patient, we should always try to ascertain whether the *Anlage* responsible for the psychosis can be considered as a purely cerebral one, or whether there is sufficient reason to assume the co-operation of an extracerebral constitutional factor.

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Enebuske and confirmed by myself, of Guttmann's and Sargant's interesting benzedrine experiments, of the not infrequent occurrence of fainting fits and delirious episodes in perfectly straightforward depressive cases, etc., etc. It would, however, lead us much too far to enter into a detailed discussion of all these still very obscure problems.

So I must once again apologize for not going deeper into these problems. If I look back on what I have said before, I see that similar excuses are only too frequent in this paper. The field to cover is in fact so enormous that I could not hope to achieve more than a very cursory review of the problems. If I have at least succeeded in showing that these problems are worth attacking, the aim of this paper will certainly have been attained.

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PSYCHOLOGICAL IMPLICATIONS OF FUNCTIONAL CIRCULATORY DISORDER.*

By E. GUTTMANN, M.D.Munich.

I HAVE been asked to speak to you about the psychological implications of functional circulatory disorder. Unfortunately there is no generally recognized system of the functional disturbances of the circulation. For this reason, and also for want of time, I shall be able to deal only with a few sections of the problem.

The functional disorders of circulation are generally classified according to the organ which they concern; their correlations within the total organism have hardly ever been studied in full. Vasomotor neuroses are known, but the vasomotor neurosis, as an entity, is only spoken of if its constitutional basis is discussed, e.g., in genetics or in work concerned with morphological anomalies of the capillaries (capillary microscopy). The inter-correlation of abnormalities of function between various vascular provinces has been investigated very little. Therefore it is practically impossible to draw any definite conclusions from observations of vascular function at the periphery as to what happens, for instance, in the brain vessels. If, therefore, the vasomotor neuroses with psychological symptoms are to be discussed, it can be assumed only with all possible reserve that something similar in kind to what can be seen in the peripheral vessels may occur in the brain, viz., vasomotor disturbances of some description. This, however, seems justified as a working hypothesis, considering the general medical tendency to attribute co-existing symptoms to one cause as long as there is no evidence to the contrary.

It is proposed to take as an example of the vasomotor neuroses Raynaud's disease, or better Raynaud's syndrome, i.e., paroxysmal local syncope, which may, or may not, lead to gangrene. There are a number of cases on record in which this illness was accompanied by mental symptoms. Heuck has calculated that $7\frac{1}{2}\%$ of all cases show mental disorder. On looking through the cases published, with a view to finding out if, and how far, the cause or the mechanism of Raynaud's syndrome may be made use of to explain the pathology of the mental symptoms, it will be found that the psychiatric findings are well in keeping with Krapf's views. There have been found confusional states (Bland, Pechkranz) and depressive phases (Ritti, Macpherson,

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Shaw and Targowla, Nedopil, Heuck) coinciding in time with the vasomotor attacks; in the case of which we have the best description (Heuck) there were also hysterical fits.

The fits accompanying vasomotor disturbances point to another aspect of the problem. Theoretically there are three ways of explaining this coincidence: (1) The hysterical symptoms are psychologically connected with the vasomotor crises, that is to say, the patient is aware of the physical symptoms and responds to this awareness in an emotional or tendentious way; (2) an emotion produces a hysterical reaction, and, at the same time, a vasomotor attack in the *locus minoris resistentiæ*; (3) similar or identical vascular crises co-occur in the extremities and in the brain, the latter producing fits. These are either (*a*) cerebral attacks *sensu strictiori* which only appear similar to hysterical ones, or (*b*) they disturb consciousness and lower the mental level to one on which hysteriform primitive reactions occur. Instead of testing the three possible modes of origin in Heuck's case, which has not been examined and described from this angle, it is proposed to demonstrate in other examples that all the possibilities outlined theoretically occur in reality.

(1) The first variant, the psychological explanation, appears to be the rarest one. In severe cases of Raynaud's and similar syndromes, the examiner is struck by the patient's remarkable capacity for putting up with the disease. It is true that the awareness of the mutilations often produces the emotions which one would expect, but gross emotional symptoms are rarely met with. Only occasionally neurotic mechanisms make use of these physical symptoms, and claims for compensation or other demands of similar psychological structure are based on them.

(2) The classical, one may call it the experimental example, of the co-occurrence of hysterical dissociation and vasomotor reaction are those cases in which the clinical picture of Raynaud's syndrome, or an intermittent local syncope, may be produced in hypnosis by suggesting emotional experiences. Such cases of so-called hysterical pseudo-Raynaud are numerous in the literature of some decades ago, when definition and differential diagnosis of this disease entity were discussed. It is interesting to note how this problem was settled by shifting the view-point: the patho-physiological (instead of the nosological) approach reveals the identical mechanism, which may be set going in various ways.

(3*a*) In order to discuss this group, organic discharges of cerebral vascular origin, it must be kept in mind, that there are cases of epilepsy in which the attacks start not only with paræsthesiæ, but even with true vasoneurotic pictures, such as "doigts morts". Furthermore, one should not lightly discard the possibility of mistaking epileptic fits for hysterical ones and *vice versa*. The recently growing interest in the epileptic fit as such has enabled investigators to see how frequently atypical motor discharges can be observed in definitely organic epileptic states, which, therefore, may be regarded mistakenly

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as hysterical. Fits with rotation around the logitudinal axis, running movements, grasping and similar automatisms, alterations of typical discharges in the various stages of dissolution of the normal motor functions under the influence of posture and righting reflexes may only be mentioned here.

The question, however, arises—particularly from the patho-physiological standpoint—how far psychogenic discharges may use the same mechanism as organic ones, and how far they really and necessarily do so; in other words, how far eventually the differences disappear owing to a final common pathway. In a particular group of the so-called affect epilepsies I have been able to demonstrate that this may be so.

(3*b*) Disturbances of consciousness produced by vascular disturbances in the brain, with consecutive hysterical reactions, can be discussed best in the light of psychoses accompanying migraine. Migraine is sometimes combined with Raynaud's syndrome. I saw recently at the Maudsley Hospital a woman who had been suffering from severe attacks of migraine for many years. She had all the typical symptoms, such as scintillating scotoma, fortification figures, unilateral headache, nausea or vomiting and post-paroxysmal euphoria. Besides, she described most clearly how these symptoms were preceded by tingling and numbness in the tips of her fingers, then blanching of the fingers followed without pain. This patient had one unusual symptom; she complained that she lost her eyesight during these attacks. Since she apparently did not behave as if she were blind, she was regarded as hysterical. No attack was observed, therefore nothing can be said about type and intensity of the alteration of her consciousness during the attacks. That there was some can hardly be doubted from her vague and patchy recollection. Considering the whole setting of the case, this alteration of consciousness must be regarded as the basis of the hysteriform reaction.

Most impressive examples of mental disorder in connection with *migraine* have been collected by Mingazzini and his school. Apart from depressive states and attacks of catatoniform excitement, they observed psychoses of short duration characterized by obtrusive, complex, scene-like, visual and auditory hallucinations. The patients saw first the usual flashes, dots and zigzag lines; from these developed figures of dead persons, black men; wild animals, barking dogs leapt on them; men armed with knives attacked them and threatened to cut their legs, etc. During these states consciousness was definitely disturbed; recollection was vague and patchy. It would be superfluous to suggest any other explanation of the confusional-delirious state than its common causation with the migrainous attacks. This must be so, however hysterical these hallucinatory episodes appear to be, with their scene-like character, their obtrusive symbolism, etc. Their close relation to the physical mechanism is further underlined by their abrupt cessation with the end of the migrainous attack.

Fainting may be discussed as a different type of vasomotor disturbance,

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without going into the question of which particular type of vascular dysfunction underlies either of them. The psychological implications are of little interest, as there is a rapid onset of unconsciousness with loss of muscle tone; occasionally if the beginning is not quite so acute, something about the dissolution of sensation, e.g., of visual perception, may be learned from self-description. Instead I should like to discuss another state in the origin of which a reflex change of the tone of the cerebral vessels plays a part, viz., the *knock-out in boxing*. It hardly needs emphasizing that this technical term does not imply any medical uniformity. I shall not speak about the psychological accompaniments of the rare reflexes from the carotid sinus or the solar plexus, but I will only refer to the typical knock-out. By analysis of observation and self-description of knock-out after punches to the jaw, various mechanisms can be demonstrated as being of importance. Either singly or by interaction they bring about the state of unconsciousness called "knock-out". Direct mechanical action on medullary and di- or mesencephalic centres and the labyrinthine organ and reflexes from the vestibular apparatus seem to be the most important factors. The latter act either on the muscle tone or the vascular system, producing a fall of the blood-pressure. Furthermore, direct damage to the vessels may occur, as hardly needs mentioning. But it seems worth emphasizing that late effects can be observed which can only be explained by functional vascular disturbances due to a disturbance in the vessel tone following the trauma. I saw lately a boxer who developed a progressive hemiplegia a few days after severe blows to his temple, whilst in the interval only headaches and dizziness were present. This point is mentioned here only because one of the numerous psychological features of the knock-out seems to be closely related to functional-vascular disturbances, viz., the amnesia without fall or unconsciousness. This is what happens: the pugilist is hit at some moment; he does not fall nor go down, but keeps on boxing almost or apparently quite normally. Retrospectively, however, he reports a shorter or longer gap in his memory which often extends from the injury backwards in time (retrograde amnesia), and sometimes forwards to cover the end of the fight or even a period after it (anterograde amnesia).

There is evidence that this is not a hysterical repression of an unpleasant experience, since the same occurs after victories as well as after defeats. Furthermore, onset and end of, behaviour during and retrospective attitude towards the amnesia are entirely unhysterical. What really exists is a post-traumatic state of altered consciousness, very similar to an epileptic twilight state. If you remember what Dr. Krapf has said about the pathology of the twilight states, you will recognize this similarity as an additional argument in favour of its vascular origin. This assumption is furthermore supported by the occurrence of similar states of changed consciousness in patients with arterial hypertension, in whom the organic nature of the condition is sometimes proved by residual focal symptoms.

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J. Ch. D—, a bus conductor, aged 53, was sent to the Maudsley Hospital because he was suffering from attacks in which his legs became stiff. He also noticed that sometimes he lost control of his right hand and so was unable to punch the tickets correctly. He had always been rather quiet, but easy-going, generally happy, steady in mood, energetic, and had always plenty of friends. For the last two years he had gradually become moody and depressed and had lost interest. He had also shown increasing difficulty in understanding what was said to him.

He had " brain fever " when at school, and an accident twenty years ago. He has been married happily for thirty-six years and has three children.

On admission he had a blood-pressure of 150/100, the deep reflexes were very weak, the peripheral arteries (including those of the fundi) were thickened, otherwise he showed nothing abnormal physically.

He was co-operative and cheerful, and behaved normally ; in performing the usual intelligence tests he was somewhat slow, but showed no marked impairment of memory (apart from the amnesia after the attacks), nor any other gross signs of dementia.

From his description it transpired that he had an amnesia for a period of some length after what he called his attacks. He could state, for example, if questioned, at what point on his route he felt the disturbance in his head, and afterwards his memory was blank until he found himself at the terminus during the usual rest period. His wife also described another type of attack in which he behaved in an extraordinary manner, banging doors and rushing up and down stairs.

While he was in hospital several attacks were observed in which he jumped about, flung out his arms and legs periodically, and rolled on the floor. He did not lose consciousness, but was not quite clear when seen immediately afterwards. His attention was difficult to arouse ; questions had to be asked repeatedly, and he fiddled with objects in an absent-minded way.

The following patient was admitted because of a hysterical outburst. Several attacks of undoubted hysterical character were observed in hospital. They could be induced by talking of the onset of her illness, or by over-breathing, or by any minor examination or test. In these attacks she shook all over and screamed. She was not unconscious, did not bite her tongue nor wet her clothing.

Miss A. F—, aged 42. Nothing noteworthy in the family history. No early neurotic traits. Standard VI in school. In domestic service since 15 years of age. She had been suffering from occasional diffuse headaches ever since she was 16. During the last three years she had had attacks of " bilious " bilateral headaches, which were strong enough to wake her up during the night.

She was described as a reserved, rather self-conscious, obstinate, cheerful, active and entirely undemonstrative personality.

She had the first attack when she was at work in the kitchen ; she suddenly felt a stabbing pain on the top of her head. She lost consciousness, and was found lying on the floor. She was not paralysed, but very weak all over.

Physically : Her blood-pressure was 230/155 mm. Hg., and rose during the attacks by 10–30 mm.

The attacks were observed to come on while the patient was asleep.

Again, it seems hardly conceivable that these particular attacks can have been of purely psychogenic origin, if one considers the previous history, the absence of emotional causes, the occurrence during sleep and the character

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of the first attack. One will have to assume that the primary cause is an impairment of the brain function due to a circulatory disturbance leading to alteration of consciousness, which provides the background for a primitive hysteriform reaction.

The main elements in these attacks in both cases were probably, in the first place, the disturbance of consciousness brought about by a fluctuation in the blood-pressure, secondly a motor discharge on an intermediate level consisting of repetitive pseudo-spontaneous movements, and thirdly, possibly an elaboration or continuation of these movements in the stage of transition to normal consciousness.

Mentioning these observations I have trespassed to some extent on Dr. Mayer-Gross's topic, since he is going to deal with hyperpiesia. However, the conception of hypertension, as brought forward by Dr. Krapf, puts it into close relationship to the vasomotor neuroses. It is, so to speak, a vasomotor neurosis on a high blood-pressure level.

It is only a short step now to raise the question whether similar clinical pictures, observed in neurotics, correspond to similar happenings in the vascular system, only on a normal level. There is very little exact knowledge which would enable one to answer this question. We know, however, that there are constitutional relationships between the vaso-labile and the hypertensive patient. Furthermore, there is material to show that vaso-neurotics have a tendency to develop hypertension, and finally, experiences of the R.A.F. in recruiting appear to demonstrate that by excluding vaso-neurotics from the service, neurotics generally can be excluded. There still remains a wide field for further research. Only a certain percentage of neurotics exhibit a high blood-pressure. There is some evidence of correlation between the blood-pressure level and the type of neurosis (S. Barton Hall). Besides, a certain percentage of patients and normal persons show a transient rise of the blood-pressure, generally interpreted as an emotional rise due to the tension during the medical examination or other extraneous stimuli, but it may be asked why all subjects do not show this vasomotor reaction. What is the particular constitution of this selected group? Of what particular kind is the interaction of the vascular system and the mental symptoms in the particular case, considering the various possible ways outlined before?

A special instance of organ-disposition can be found in neurotics with low and labile blood-pressure—a group which may be mentioned here as a contrast and a supplement to that showing vaso-labile hypertension. Very little attention has been paid to this group from the psychological standpoint. As I see it, two types may be distinguished: one consists of asthenic, fatigable, permanently tired and easily exhausted persons, inclined to faint and to break down in neurosis; the other group inclines to create tension artificially, to breed conflicts and intrigues. A well-known type of hysteric seems to belong to this group. Also the inclination for some blood-pressure-raising drugs,

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such as caffeine or benzedrine, may be rooted in this constitutional predisposition, whereas the vasodilator alcohol is the favourite drug of the pyknic who tends to hypertension.

A few words should be said about the psychological symptoms in functional heart disease. The general trend of investigation should be again to study the psychological accompaniments of well-known disturbances of the heart function, in order to learn finally what anomalies of function may be expected to be found as the basis of the neurotic symptoms of which the patients complain. There is little reliable material available on which to base our views. Fahrenkamp has systematically collected interesting material which demonstrates a remarkable discrepancy between patients with organic and functional heart trouble concerning their subjective symptoms and their attitude towards illnesses, or work, or pleasure. Roughly it seems that those patients who complain verbosely about sensations in the heart, about palpitations and anomalies of rhythm, who feel largely interfered with in their capacity for working, and who live more or less the life of an invalid, show no clinical signs of structural change or incompetence of the heart. On the other hand, it is striking how little patients with heart failure and decompensation complain. These patients often only come to see a doctor when urged by their relatives, and it is often hard to make them adapt their work and pleasure to their state. "The psychological problem is not solved," says Fahrenkamp, "as to why the self-confidence of patients with severe heart disease is so little impaired and why these patients have such a non-neurotic attitude."

This problem indicates one line of psychiatric research in *organic* heart conditions, whereas in cardiac neurosis "the choice of organ" is the point of conspicuous interest, in other words, the question, How far is it the symbolic expressive significance of the heart, how far is it some alteration of its function that determines the neurotic to be a *cardiac* neurotic?

A promising mode of procedure seems to me to find out whether, and if so, which, anomalies of function are met with in these neuroses, how they may be influenced, and how their alteration changes the picture of the neurosis or possibly of the neurotic personality.

The most impressive psychological symptom of a disturbance of heart function is the "vital" anxiety, the fear of death or "impending dissolution" in angina pectoris. Many hypotheses on the physiology of anxiety generally are based upon cardiac anxiety in angina or the so-called pseudo-angina. There is a serious objection, however, to this view, in the fact that *pain*, not anxiety, is regarded as the cardinal symptom of angina by the most experienced writers. Paul D. White, for example, says, "There may or may not be fear of death; in my experience such fear is uncommon; certainly it is absent in the majority of cases". Sir James Mackenzie's view was similar. He stated: "Apart from pain, other symptoms may be present, such as constriction across

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the chest, a sense of great depression." On the other hand, the same author said: "There are a number of patients who have sudden attacks of great oppression across the chest, a sensation of sinking, and of impending dissolution, in whom there is no pain. . . . I am disposed to think that in some the attacks are brought on by the sudden onset of an abnormal rhythm, such as auricular fibrillation or flutter."

Furthermore, theories which take cardiac anxiety as the starting-point for the explanation of anxiety in general have often not taken into account that identical or similar sensations may be elicited from other sources as well, and that even in heart conditions there is often not a localized sensation in the heart, or in the chest, but a general feeling of suffocation. Studies of physiologists (Haldane) and clinicians (Wassermann) have shown that probably the oxygen need of the tissues produces those sensations which are in the centre of the experience "anxiety", and it is interesting to note in this connection that attacks of angina pectoris can be induced in favourable subjects by rebreathing low oxygen mixtures (Carl J. Wiggers).

Dattner has tried to explain neurotic anxiety as due to insufficient oxygen supply to the tissues; but experimental proof of this working hypothesis is still scanty. In patients with a particular neurotic picture, the so-called psychogenic hyperventilation tetany, I have been able to show some relationship between tetanoid hyperexcitability (demonstrated by measuring the galvanic threshold of the peripheral nerves) and the predisposition for anxiety attacks, consecutive overbreathing and fits. Considering the influence of alkalosis on the capacity of hæmoglobin to supply oxygen, one can interpret these findings in the light of Dattner's views. Such analyses of small clinical groups appear to me more promising than premature generalization and theorizing.

Many problems which arise in studies of "anxiety" in its psycho-physiological aspect seem to be due to poor definition of the terms used. For many psychological facts are mixed up and summarized under the clinical term "anxiety". Fear, the recognition of and reaction to danger, has often been contrasted with anxiety as objectless fear. This antinomy, however, does not exhaust the problem in question. The objective incapacity for handling a situation and the reaction to this incapacity—called the "catastrophe reaction"—are, according to Goldstein, the deepest roots of "anxiety". On the other hand, according to Jaspers, asking oneself questions about the fundamental metaphysical problems (such as Why am I? Why is something? Why is not nothing?) will cause an experience of "anxiety". For emotions of this kind one can hardly expect the same physiological substratum as for the sensation which one experiences after running up a staircase too quickly.

Therefore the first demand is for a detailed analysis of the psychopathology of anxiety. Only this will furnish the basis for investigation into the pathology and patho-physiology of the various types.

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I wish to express my gratitude to Prof. E. Mapother for his kind permission to use the clinical material of the Maudsley Hospital.

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PSYCHOSES ASSOCIATED WITH HYPERTENSION, ARTERIOSCLEROSIS AND HEART-FAILURE.*

By W. MAYER-GROSS, M.D.Heidelberg.

THE clinician's share in a discussion on psychoses of circulatory origin must be to provide, in the first place, a description of the characteristic clinical pictures. This would mean the enumeration of a rather puzzling variety of observations, among which it has until lately been rather difficult to find any systematic order. One could scarcely add very much to the classical chapter on arteriosclerotic psychoses in Kraepelin's text-book. Since, however, Dr. Krapf has singled out the hypertensive psychoses as a special group, the task is no longer one of pure description. The application of the achievements of general medicine to the field of hyperpiesis should have opened new aspects in cerebral vascular disease. Supplementing the fundamental ideas given in Dr. Krapf's own paper, I propose to give you a more detailed survey, with some illustrations.

There are two main types of psychotic symptoms in which a close association between increase of blood-pressure and psychosis has been established; to one he gives the name of *twilight states*; the other is characterized by *episodic emotional fluctuations*. The twilight states are a rather varied group, but have the common feature of a more or less severe dimming of consciousness. Some show confusion and delirious symptoms, others are of catatonic character, others show only a state of panic with impulsive actions—always combined with some obnubilation. The emotional changes are mostly depressive, often with some anxiety, or of a paranoid kind with increased irritability.

The hypertensive twilight state is furthermore characterized by its sudden onset. It starts overnight, like an apoplectic stroke, often beginning with a fainting attack. There are prodromata which resemble those preceding a real apoplexy. The twilight state may be precipitated by all the well-known events which can release an apoplectic stroke. Its duration is short, between four and twenty days in the typical cases; death may occur, caused by the underlying circulatory breakdown; but if the patient survives, complete restoration of function may be expected.

Emotional fluctuations associated with hypertension are sometimes so transient and brief that they are never seen by the psychiatrist; in other cases

* Expanded from a paper read at the Annual Meeting of the Royal Medico-Psychological Association at Ayr, July 8, 1937.

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they may last over weeks and months. Again the outlook of such affective disturbances is either restitution or a fatal end.

Mixtures of these two typical forms of hypertensive psychosis are common. On a depressive state of longer duration, one or several shorter periods of dimmed consciousness with confusion or excitement may be superimposed. These mixtures seem to me even more characteristic than the pure forms. Repeated control of the blood-pressure should never be missed if one comes across such a combination in middle-aged patients.

At the margin of the two main types Dr. Krapf found *epileptiform symptoms*, especially absences, which frequently occur in the prodromal phase and are often overlooked.

Finally, the *changes of personality* associated with hypertension are rather characteristic, and clearly distinguishable from what one finds in arteriosclerotic and senile disease. The patient loses his elasticity and adaptability in regard to all mental processes. The positive symptoms he exhibits are an unveiling or an exaggeration of psychological features which were present before, either latent or less manifest. There is no definite decline of personality, and the changes are potentially reparable. Under the pressure of an emergency the patient may—for a short time—appear his former self, entirely unimpaired.

Looking back on these syndromes and symptoms associated with hypertension, one sees that they cannot be summed up under one of the "reaction types" or other symptomatological units. This is just what one would expect if the functional disturbance of circulation can affect smaller or larger parts of the brain or different regions to a different degree.

The following cases, taken from a larger material collected in the Maudsley Hospital, may illustrate the principal types of symptoms and their combinations. The first case shows twilight states of short duration, with paranoid content scattered over a period of several months. This is followed by a kind of agitated depression with marked anxiety, but without paranoid symptoms.

CASE 1.—Mrs. C. V—, aged 51, was the eldest of eleven children of a father who was a heavy drinker. One sister suffered from "fits". The mother died early from unsuccessful operation for brain abscess and the patient had to care for her younger brothers and sisters; she was a poor scholar. Her first husband, whom she married at 19, deserted her, leaving her with two children. She married again a much younger man, and had another daughter, now aged 7½. She was an active and lively personality, sociable, somewhat hasty tempered. There was nothing noteworthy in the medical history.

About Christmas, 1934, she complained of swelling in the neck. She became tremulous and shaky, could scarcely stand up and did not go out for four months. She was depressed and became "delirious" for a few hours every few days. She then stared blankly at the ceiling or chanted hymns, oblivious of her surroundings. Her face was flushed. She expressed on such occasions paranoid ideas; her relations were talking about her and wanting to "put her away". Between the attacks she never expressed such ideas.

In November of the same year, four weeks before the beginning of her illness, Mrs. V— had fallen off a tram. Although this did not affect her at the time,

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the husband related her illness to the accident and asked for compensation. An action was still pending during her stay in the Maudsley Hospital.

She was admitted here on June 25, 1935, referred from a general hospital, where she was diagnosed as neurasthenic, and high blood-pressure was noted (190 120). Her general behaviour in the ward was pleasant and normal apart from anxiety and restlessness. In talking to the physician she was self-effacing and apologetic. She continually worried about herself and asked for reassurance. She was a little overactive, and sometimes seemed almost unable to control her agitation. She realized this herself and complained about it, besides various other complaints. Among these the feeling of needles and pins in the three outer fingers of her left hand seems noteworthy; no sensory anomaly to tests was found in this region. She denied all the delusional ideas reported in her history; memory and intelligence were not impaired. During her two months of treatment in hospital no confusional or delirious state was observed. The patient improved, became much quieter and lost her fear of traffic.

She left the hospital rather suddenly (August 19, 1935), but afterwards attended the Out-Patient Department. Her state varied a good deal. She was much better in the first half of 1936, but still had numerous complaints, similar to those during in-patient treatment, when she attended for the last time in September, 1936.

Physically, generally well preserved, some tremor of hands, slight swelling of the thyroid on admission, but no tachycardia and normal impedance angle. Blood-pressure 180 100, fluctuating down to 155/85; at the time of discharge the latter was the average measurement. No abnormal findings in the heart. No neurological findings.

The following history may be taken as an example of a depressive illness with paranoid features in a case of hypertension. Epileptic fits and a twilight state occurred rather unexpectedly during the agitated depression without very much influence on its course. The outbreak of the psychosis a few months after thyroidectomy seems noteworthy.

CASE 2.—Mrs. V. M—, aged 41. Mother and sister suffered from exophthalmic goitre; father was an active, successful business man, who died at 62 of a stroke. Patient was a sensitive child, without neurotic traits; did well at school, and afterwards lived for five years at home; fond of all sorts of social entertainments. She worked as a nurse during the war and married in 1921. Her personality was described as energetic, capable and lively; she was very fond of music, liked to play a leading part in parties, rather extravagant in her clothes. She has one healthy son, but avoided further pregnancies, as she was afraid of the goitre. In 1925 she was ill with pleurisy and septicæmia; infected tonsils were operated on in 1928, after which she felt especially well. Signs of Graves's disease were present since 1916; she had iodine treatment for over ten years. In 1933 she complained of choking in her throat; she saw Sir Thomas Dunhill, and thyroidectomy was performed in May. She made a good recovery, but seemed rather restless and irritable. In September, 1933, this increased to a nervousness and excitability, regarded as morbid. Depressive and paranoid ideas came on in attacks; in the intervals she seemed quite normal. She lost weight (1½ st. since July, 1933). In January, 1934, she became more suspicious, refused her food and was self-reproachful. One day at breakfast, as she was left alone for a few minutes, she locked the door and grasped the burner of an electric stove. She was immediately discovered but had severe burns on both hands. Three weeks later (February 22, 1934) she was admitted to the Maudsley Hospital in a state of agitated depression, with attacks of anxiety. She talked ceaselessly in a wailing tone, accusing herself of all kinds of wickedness. She answered questions coherently and to the point, but always returned to her self-reproachful ideas: she should be sent to prison

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and will be hanged, etc. She speaks of the unreality of the outer world. No disturbance of memory or intelligence. This state of restlessness with extreme depression lasted nearly all the time she was treated here. She seemed sometimes calmer, but shortly afterwards was again agitated, trying to hurt herself by knocking her head against the wall, etc.

On April 4 she had a series of five typical epileptic seizures, after which she had an extensor plantar response on both sides. Afterwards she was confused and disorientated and complained of frontal headache for some days. On April 11 the confusion had disappeared and she showed again her former depressive picture. Another fit occurred on April 19. Somnifaine treatment applied in June had no effect. From time to time she removed the bandage from her hands, and large amounts of sedatives were needed to manage her in a quiet ward. She was transferred to Bethlem Hospital on July 4, 1934.

The blood-pressure was 180/120 on admission; further readings were: February 26, 1934, 165/100; April 5, 1934 (day after series of fits), 120/85; April 13, 200/120; April 17, 205/120; April 19, 185/115; April 23, 175/120; June 4, 180/110; later about 200. No abnormal findings in the heart. No neurological findings apart from the fits.

Two patients with so-called involutional depression may illustrate the variety of affective psychoses associated with hypertension. In the first case transient symptoms of vestibular impairment and disturbances of other cranial nerves (tic, stammer) are of some interest. The patient made a good recovery after three months. Both the parents of the other patient suffered from circulatory illnesses. The clinical picture is an interesting mixture of organic and affective symptoms.

CASE 3.—Miss W. M. T—, aged 42, nursery governess, daughter of a minister, comes from a generally healthy family. One of her sisters was very nervous as a child and so was the patient. She suffered from nightmares and fear of the dark, and walked and talked in her sleep. She was a good scholar, and had a Froebel training after leaving school. She worked successfully as a nursery governess with difficult children and liked this kind of work very much. She was pleasant, energetic, capable and respected for her high ideals. To herself she was always timid, over-scrupulous and over-conscientious. She had masturbated since early youth, and always felt guilty and self-reproachful. She could deal with masturbating children sensibly and successfully, but was unable to cure herself of the habit.

She had her first attack of rheumatism at 11 years of age, and several relapses since. Since 1932 she had had repeated attacks, had had all her teeth removed and been treated in several hospitals. After ten weeks' treatment in hospital she went to the seaside in March, 1935; the doctor there encouraged her to do exercises. After returning home her nervous trouble started; she had headache, pains in the limbs, suicidal ideas and increased worry over masturbation. A slight stammer and a facial tic, which she had never had before, developed.

On admission to the Maudsley Hospital (July 1, 1935) she was anxious and very much concerned with her health. She complained of irritability, hypersensibility to noises and sleeplessness. She had the subjective feeling that her bed was heaving about and rocking. She was frank and co-operative, and gave an intelligent account of herself. She willingly accepted reassurance with regard to her worries and showed relatively good insight into her condition. There was no retardation; memory and intellectual functions were entirely unimpaired. Over a considerable period of the treatment in hospital she felt tired, mentally exhausted and depressed. After a month the stammer and facial tic had improved. Sometimes the clinical picture was that of hypochondriasis with changing complaints of all kinds of

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ailments. Towards the end of the third month she became more cheerful and less self-centred. At the end of October she left the hospital very much improved, but still slightly worried about work prospects.

Besides the facial tic the bodily findings were: Pulse regular, full and high tension, nothing abnormal in the heart apart from a loud second aortic sound. Blood-pressure was 230/120 on admission and fluctuated between that and 190/130; at the time of discharge it was rather constant at about 190/120. No neurological findings. Fundi normal.

CASE 4.—Mrs. B. E. P—, aged 51, is the daughter of a drunkard, who died at the age of 51 of "paralysis of the brain" and Bright's disease. Her mother suffers from heart trouble. Most members of the family are described as highly strung and irritable; one sister is at present being treated for depression. Mrs. P— was a healthy child, quite good at school and popular with her schoolmates. She went into domestic service; worked as a ticket collector during the war. She met her first husband in a hat factory where she afterwards worked. He was a drinker; they separated after four years and patient became a waitress; as such she has worked since, always with the same manageress. There is one child, aged 12, described as very nervous. Patient's personality was characterized as sensitive, over-conscientious, excessively orderly, independent, successful in her work, sociable with employers and workmates. Her mood was generally cheerful; at one time she had the nickname "the smiler".

When the child was born she suffered from swollen legs. For the last five years she had suffered from gastro-enteritis and was rather concerned about her health. She never felt quite the same since the stomach trouble began. Her energy seemed diminished; her doctor diagnosed "neurasthenia".

Menopause started five to six months ago; she suffered from flushings and headaches, and a depressive illness became more and more marked. She had swollen legs and feet; was unable to do her work or concentrate. Her movements and speech became slow; she lost interest in life, was suspicious and full of hypochondriacal worries. She had the feeling that everything was in a muddle; she could not taste her food and sometimes felt sick.

On admission (November 26, 1936) she looked cyanosed and had gross œdema of both legs. Pulse 60, high tension, full volume; cardiac dullness enlarged, aortic sounds sharp. Blood-pressure 186/108. Quite marked general wasting.

She was retarded, very depressed, seemed absent-minded, and answered doubtfully if questioned about orientation. At the first interview she talked coherently, but was very slow in movement and in answering. In the case of dates especially it took a long time before she found the right answer. She seemed very suspicious, resented everything the doctor ordered and complained about her bowels. "It seems wicked to eat food and not to be able to taste it." "I am in a whirl; I feel a bit dizzy; I can't concentrate—I can't make up my mind." She often gave the impression that she could not find the right word to express herself and seemed very distressed in consequence. Retardation became less during the following weeks. Sometimes the patient cried bitterly and was full of self-reproach and rather restless. There was often very little depressive expression, and she behaved more like a child that does not get what she wants. She had attacks of confusion at night in which she was completely disorientated and repeated questions in a perseverating manner. During the daytime she was sometimes overcome by a sudden anxiety.

Since May she has become more quiet, remains in bed, is resistive and inaccessible, says hardly anything that can be understood, although she continuously mutters in a whispering tone. Her facial expression is a fixed, depressive one. Sometimes she uses abusive language to nurses who try to feed her.

Blood-pressure readings, August 3, 1937: 10.15 a.m., 120/90; 11.15 a.m., 110/80; 12.15 p.m., 120/90; 1.15 p.m., 150/90 (after having been fed against considerable resistance); 2.15 p.m., 145/95; 7.20 p.m., 130/90.

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The last case is given as an example of the personality changes in hypertensive patients. The descriptions obtained from her employer are very characteristic and need no commentary. A slight episodic depression is still present.

CASE 5.—Miss J. W—, aged 46, comes from a healthy family. Nothing abnormal is known in her early development. She was good at school, got on well with other children, but never made friends. At 14 she started work as a clerk in a large chemist's firm, in which she was employed for 32 years until her present illness began. She worked hard, took evening classes in shorthand, etc., often three times a week. She was conscientious, ambitious and methodical, always punctual and interested in her work. For years she lived with her mother, who suffers from chronic arthritis. In her twenties she was once engaged to a young man, who died suddenly. She was never a "good mixer", had no hobbies and no special interests. Her mother describes her as usually cheerful, but irritable when tired. She speaks of herself as pessimistic and anxious, and of rather a worrying disposition. Apart from attacks of influenza she was never physically ill. A first "nervous breakdown" occurred after her father's death in 1925. She felt unable to concentrate. After a month's holiday at the seaside she recovered completely. In 1932, after the resignation of her chief, she became upset by the reorganization of her work; she had similar complaints to those of 1925. This time a holiday of three weeks' duration led to recovery.

After the retirement of her chief, patient became much more difficult to handle according to the report of her superior. He gave a characteristic description of this development. She had always been slow in her work, but became much slower. She bitterly resented all changes in method. She had always done this, but now she felt unable to cope with her new work, and argued and made a thousand excuses. When she was moved to another department she grumbled because she had no shorthand to do; when her chief gave her letters, to keep up her shorthand, she complained she had no time to do them. She never had been able to stand any interruption; but now, if she was asked to leave off what she was doing to do another job for ten minutes, she would make an endless fuss. She had always been sensitive to criticism, but in the last few months she was liable to become furious and sometimes definitely rude. With regard to her workmates she seemed suspicious and unco-operative, giving her confidence to no one. Nobody dared ask for her help with any extra work, and if they offered to help her in their turn, their offer was received very ungraciously. She took everything as a personal offence; at times she left the office without saying anything, and the manager had to send a telegram to ask what she had done with the keys. All these difficulties were present in a small way beforehand, but had increased within the last years, although everyone in the office had been most forbearing with her.

About Christmas, 1934, she had an attack of influenza, from which she was unable to recover. Twice she went to seaside places for a week or two, but did not feel any better; she resumed her work for three days, but felt too tired and listless to carry on. For weeks she stayed at home, mildly depressed and with a complete lack of energy and interest in her surroundings. After seeing several physicians she was admitted to the Maudsley Hospital April 1, 1937.

Since then her mental state has slowly improved, but remained the same in principle; she was retarded, sometimes rather lethargic and without concentration. She tried to co-operate in interviews, but her answers were slow and monosyllabic. There were only a few complaints: depression, lack of energy, feeling of being tired, loss of appetite. Memory and intelligence were entirely unimpaired. After weakly resisting for a time she consented to being pensioned by her firm. She never showed very much depressive affect, and lives quietly and apparently contentedly the routine life of the hospital. She becomes tearful if spoken to and a certain amount of retardation is still present.

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There are no abnormal physical findings apart from the blood-pressure. Some of the readings were: April 1, 1937, 190/100, 200/110; April 10, 180/105; May 14, 180/105; ten minutes later, after bad news, 155/105; July 20, 145/105; August 3, 11 a.m., 150/190, 12.15 p.m., 155/110, 1.15 p.m., 160/80.

Reparability of function brings the hypertensive cases into marked contrast with the arteriosclerotics. If one tries to sum up the characteristics of the pure arteriosclerotics in rough outline, one can speak of a slowly progressing deterioration of the personality with more or less marked focal symptoms; there are no twilight states and no lively fluctuations of mood in pure arteriosclerosis; the whole clinical picture is shallow and indistinct as compared with hypertensive illness. Decline of emotional response, fading of interest, intelligence and memory go parallel and are more or less definite.

Such arteriosclerotic mental changes may follow an hypertensive phase, or both may occur in combination (Case 4 ?). Differential diagnosis, therefore, may be difficult in the individual case. Nevertheless the general characterization and separation of the hypertensive group by Dr. Krapf seems to me a progressive step. It is very much in favour of his differentiation that it is in accordance with pathological findings and with the discriminations made by general medicine in the field of arterial disease.

From the newly-gained viewpoint it was a matter of course that one should try to penetrate what Kraepelin had called the darkest chapter in psychiatry—the *presenile psychoses*. It would be premature to draw definite conclusions from what scanty material has been sufficiently examined with regard to blood-pressure fluctuations. Still, it seems worth while to do such examinations, and it should be realized that in acute psychoses of middle-aged people vasomotor dysregulation in the brain plays an important part.

Is this part really causative, or are we dealing with an intermediate link in a causative chain which we are al le to grasp on account of its peculiar susceptibility to measurement? Even if the latter should prove true, its value in nosological explanation would remain great enough. There is another difficulty to deal with—the existence of *negative cases*. Long-standing severe hypertension can be present, without any psychic disturbances, at least without manifest psychosis; very marked arteriosclerotic changes in the brain vessels are found in people with excellent intellectual and personal preservation.

Factors of *time* and *intensity* may explain some of these incongruities. A slowly developing arterial illness to which the brain is able to adapt itself may have little or no effect on its function, whereas more or less sudden interference with cerebral circulation will probably produce strikingly morbid responses. It is obvious that there must be interaction of the factors of acuteness and intensity. We must admit, however, that we really know very little yet about the question whether, for instance, a relatively minute change in blood-supply of long duration has a more detrimental result than severe vascular impairment lasting over a short time—and vice versa.

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One is unfortunately not on much safer ground if one relates the different clinical pictures to the intensity of the underlying vascular process. With the exception of the most severe and general disturbances, any conclusive judgment seems as yet impossible. The reason for this is probably the interference of this factor of intensity with the local distribution of the disturbance in different parts of the brain. I shall return to this problem shortly.

I would like to speak first of some indirect ways of accounting for the variety of symptoms in cerebral vascular disease, viz., by considering all those factors which may influence the clinical appearance apart from the direct vascular effect on the brain. Influences of *constitution* and original equipment were already mentioned when I spoke of the revealing of latent character features. Sometimes it is not the exacerbation of the former personality traits, but a hysterical reaction, the tendency to which is common to all mankind, which signalizes the beginning of a cerebral vascular disintegration. I have found it quite a helpful clinical rule to consider this causation in all cases in which hysterical symptoms appear for the first time beyond the age of fifty, even if the psychological situation seemed quite sufficient to account for the patient's reaction.

There is no need to exemplify the wide range in which constitutional factors can be used for explaining unusual symptoms. Nearly everything can be understood by relating it either to the prepsychotic personality or to hereditary factors, especially if one makes use of the interchangeable correlations provided by Kretschmer's system. Such a procedure can certainly be helpful in the individual case ; but one feels rather uneasy, because one never knows how far to go back in the remote ancestry in the search for an abnormal individual, nor what trifling differences of bodily or psychic constitution among the relatives or in the patient himself might be of significance. It seems remarkable in this connection that in Dr. Krapf's material of hypertensive psychoses, no similarity between the psychological anomalies of relatives and the clinical pictures associated with hypertension was discovered.

Apart from constitution, some of the symptoms are probably connected with dispositional factors linked up with the phase of life in which the circulatory disturbances occur. The preference of the involutional and presenile age for the *melancholic* and the *paranoid* form of psychotic reaction is an empirically established fact. One knows that disturbances which are, as far as our present knowledge goes, most probably of *genetic* origin (e.g., schizophrenia) commonly assume one of these symptomatological patterns when they occur beyond the forties. No wonder that circulatory disturbances take the same appearance. A convincing explanation of this dispositional preference cannot be offered, and one feels still more helpless if questioned about the detailed peculiarities of the psychoses of the middle-aged—lack of retardation in depressions, *délire de négation*, fantastic content of paranoid ideas, etc.

Thus, naturally, one turns to the brain, which one should expect to

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contribute to the variability of circulatory psychoses owing to its high differentiation and complex organization. It is a historical fact that cerebral neurophysiology owes a large part of its knowledge to observations gained from patients with vascular diseases. History teaches, however, the not less impressive lesson that all attempts to produce systems of psychology along the same lines have failed. However wrong these systems may have been, the problem has never died down, and one cannot—I think—dispose of it by the modern commonplace that the brain always acts as a whole.

In a former paper (with E. Guttmann) I described a variety of symptoms which constitutes what is usually called *dementia* and we discussed their occurrence and interaction and their possible relations to focal lesions. Further observations of similar cases showing vascular or senile deterioration have confirmed the idea that the relation between certain psychological anomalies and more or less circumscribed cerebral regions is still a field for promising studies. These, though starting from so-called alpha-symptoms (aphasia, apraxia, acalculia, etc.), are not meant to be confined to circumscribed disabilities. There is no obstacle in principle in the way of interpreting symptoms as occurring in psychoses from the view-point of localization; it would, in fact, be easy to give examples in which such a procedure has proved to be of advantage. This, however, would lead rather far from my present topic. What I should like to emphasize is the great importance of the circumscribed circulatory disturbances in this field of research.

Mental changes in cardiac disease was the topic of a paper by Dr. Gibson, read at the Annual Meeting of this Association at Oxford in 1930. He pointed out that psychoses which are associated with heart failure, and have a probable causative relation to the cardiac condition, are rare if one considers the frequency of heart patients with more or less severe circulatory disturbances, from which cerebral circulation is certainly not immune. The observations recorded in Gibson's paper were unfortunately not investigated in regard to the findings in the brain. In 1932 Bodechtel published histopathological studies which are deserving of the special attention of clinicians. He found marked parenchymatous defects of certain cortical layers in heart cases who had never shown any gross mental anomalies.

Our knowledge of the *minor psychological accompaniments of heart trouble* in unfortunately rather scanty. The symptom of cardiac pain, so important in practice, has however for long attracted the interest of cardiologists. Quite recently it has been studied from a more psychological angle by Doris Baker and by Geoffrey Bourne. The combined investigations of Bourne and E. Wittkower have shown the value of collaboration between the cardiologist and the psychologist. The literature is still very restricted and fragmentary, as Gibson found it seven years ago. Moodiness, irritability, restlessness and anxiety are usually mentioned in textbooks as occurring

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during decompensation and in rather severe cardiac conditions. Speaking of anxiety, there is much speculation in the literature about its connection with the heart, mostly based on popular ideas and the subjective feelings of the normal. But nobody has, to my knowledge, examined the psychic states of an average group of heart patients with regard to this and other psychological problems. Such an investigation would have to consider not only the severity, but also the kind of heart disease—a point already mentioned by Gibson—and its effect on the different parts of the circulation ; it would have to make use of all means of modern cardiology and psychiatry.

Some figures are available about *anxiety in psychoses* of heart patients. Among 22 cases observed in the Berlin Clinic anxiety was only absent in 3 cases. There was either a continuous anxious mood with attacks of panic, or a delirious state coloured by marked anxiety.

States of confusion with fluctuations of consciousness are the most common clinical picture in psychoses of heart patients. These fluctuations can be very transient ; they often occur only at night, lasting for a period of less than an hour (Krapf). In some cases the interdependence between psychotic symptoms and the degree of decompensation is quite evident. In others, however, the psychosis precedes decompensation, or even starts at the time when the heart function recovers during absorption of the œdema. In the latter cases unknown toxic influences were thought to cause the mental symptoms. Similarly toxic liver products have been held responsible, on the assumption that psychoses would only occur when the liver function was affected by the cardiac failure (Stroomann). There has, however, been an increasing tendency lately to reject unknown toxins, and instead to base theories on the mechanics of vascular functions.

Thus the part played by blood-pressure fluctuations deserves special consideration also in psychoses of heart patients. The attention paid to constitutional factors has varied considerably. There was a time when an incidental cardiac illness was unconcernedly taken for the cause of a depression or epilepsy. This was followed by the utmost reluctance to make causative assumptions of this kind and an increasing tide of constitutional theories. Gibson emphasized the "specific psychological tendencies" of the patients who respond with mental symptoms to circulatory changes ; but he was not able to illustrate the character or the special nature of such tendencies. He was sceptical in respect of explanations from the cardiological point of view. The problem is certainly intricate and obscure in many respects. It seems now ripe for a new attack to be launched on it in co-operation with general medicine.

I wish to express my thanks to Prof. Mapother for his kind permission to use the clinical material of the Maudsley Hospital, and to the Rockefeller Foundation for supporting these studies.

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Discussion.

Dr. A. G. DUNCAN said he felt somewhat reluctant to open the discussion. The papers had all been very interesting. But, at the risk of exposing his ignorance, he must confess to some difficulty in forming a clear conception of what was meant by a functional disturbance of the cerebral circulation. This term "functional" used to be applied to a great many conditions, a large proportion of which were now recognized as due to a great variety of organic causes, and he asked those who this morning had used the term so freely—and on the previous day also—as a cause of circulatory disorder to explain more clearly what they meant.

With regard to the vaso-constriction which, he gathered, occurred, he asked whether it was the result of some inherent defect in the vessels, or the result of some toxin acting on the vessels. If there was a toxic cause acting on the vessels, one could not exclude a toxic cause acting on the neurones. The brain had been so formed that it was able to stand an enormous amount of insult, vascular and other, and he found great difficulty in accepting the picture of a brain which was unable to protect itself from transient vaso-constriction, even if such could be proved. He would be glad of further information on those points.

It seemed to him that mental disorder was only likely to arise if there was a very extensive and diffuse affection of a large amount of cerebral cortex. Small and transient lesions, such as could result from local ischæmia, were very unlikely to produce permanent or prolonged mental changes, and that was borne out by the fact that arterio-sclerosis was very common in people over fifty years of age, while mental disorder in them was relatively uncommon. There must be ischæmic patches in the brains of many people of sixty to seventy years of age who had never had any mental abnormality as a result. At the risk of drawing a red herring, he was inclined to attribute those mental changes which occurred after circulatory disturbances, to "constitution", but perhaps he had better not enter too far into that on the present occasion.

Dr. A. A. W. PETRIE said that there were three points he wished to touch on. He was hoping that Dr. Guttmann was going to enlarge further on the physiology and pathology of the knock-out blow in boxing. The blow about the head and jaw referred to was a good deal mixed up with concussion, and what the boxer himself remembered was influenced by the fact, common to all sport, that the individual was so concentrating on what he was doing as to have an imperfect realization of what was really happening. A much purer form of knock-out was the blow over the "solar" plexus giving sympathetic and vaso-vagal shock, and no doubt leading to various circulatory changes through stimulation of the sympathetic and parasympathetic system. This could even be produced by the individual giving himself a sharp tap with a forefinger over that area while relaxing the abdominal muscles.

In regard to anxiety states, the sudden paroxysmal attack appeared especially associated with circulatory changes, particularly in those cases in which patients complained of a bursting feeling in the head. While in a number of cases psychological causes were evident and could be dealt with, at times vaso-vagal attacks, stimulated by or associated with some local irritation seemed a factor. Sometimes the person would localize the origin of the attack to some part of the gastro-intestinal tract. Some patients developed an interesting technique with the object of getting rid of the attack; some jumped on or off buses, and some went indoors and some went out of doors, while some rubbed a particular part of the body, and numbers stated that getting hot or over-heated was a predisposing factor. This suggested some disturbance of the vascular dispositions as well as the nervous factor.

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Vascular disturbance was an important matter in regard to the Royal Air Force, and it was essential to study this because of the sudden changes of posture and position which had to be adopted in the air. For this reason, neurotics, particularly sensitive to vascular disturbance, were unsuitable material.

Lastly, there was the problem of the person admitted to hospital in a state of confusion. Sometimes it was very difficult to tell whether the case was one of senile or presenile dementia, or whether it was one of cardio-vascular disturbance which would improve with the restoration of the general circulation, by attending to the cardiac condition or when some cerebral vascular lesion had had time to subside. The history as to onset was important as a guide.

Dr. P. K. McCowan desired especially to congratulate Dr. Krapf on his work, but before accepting his suggestions he would like to question the author on one point concerning it. If these twilight states were due to hypertension rather than to arterio-sclerosis the patient should act as his own control. He asked whether Dr. Krapf had found, in taking blood-pressures regularly in those patients, that whenever they had a definite rise of blood-pressure the twilight state supervened. And did he find that by lowering the blood-pressure he was able to cut short the attack? He hoped that what Dr. Krapf said was correct, because if so it would greatly elucidate the problem and would hold out hope as to the effects of treatment.

Dr. J. H. MacDonald said he had been asked to say a word on the subject of mental disturbance in association with vascular disease. After listening to the contributions of the preceding day and this morning he felt that what had been said on the whole theme was very valuable, and he thought the Committee responsible for the selection of the subject were to be complimented on their choice.

In speaking of the vascular changes and the circulatory changes, they were considering a mechanism, rather than making an attempt to explain the ætiology. It was not sufficient to say that any mental disorder was due to vascular or circulatory change; it was necessary to get behind that and ask what was producing that change.

He had been impressed, both yesterday and to-day, by the reference to disorders of circulation which were not necessarily vascular disorders. Reference had been made to functional disorders of the circulation which caused mental changes, and of this there could be no doubt. The common faint, with total loss of consciousness, was a familiar example, and the striking instance of angina pectoris was another. Without doubt, changes in the circulation in the brain produced changes in the mental condition, and it was admitted that those might not be accompanied by vascular disease, so-called, at all.

With regard to functional conditions, the contributions had been very interesting. The question had been asked what were functional disorders—what disorders should receive that name? One had only to think of the possible change produced by the pouring out of a little extra adrenaline into the system. That was not called a vascular change, but it affected the cerebral circulation, and brought about a change of mentality.

He drew a distinction between circulatory changes and vascular changes. When speaking of vascular changes one turned to senile and pre-senile psychoses, because, whatever the ætiology, one found here mental disorders associated with vascular disease, proofs of which were so evident on examination of the cerebrum itself, in the form of softening and sclerosis.

He had listened with great pleasure to Dr. Krapf's paper, which he had found very suggestive and helpful. If Dr. Ivy Mackenzie had been present he would have made a strong point in differentiating between arterio-sclerosis and atheroma. Pathologists to-day had very well differentiated these conditions. Hyperpiesis

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was a very significant condition. The pre-senile and senile psychoses were a very mixed series of conditions; all sorts of clinical descriptions of them had been given. It was a true saying that a man was as old as his arteries; it was very well shown in pre-senile conditions. Some conditions were explicable on account of changes of the circulation, in brain changes resulting from under-nutrition. In the senile states there were definite vascular changes. Reference was continually made to focal lesions; they were really accidents occurring in the course of this disorder; and the exact clinical picture depended on what the accident was. At the visit to the engineering works on the previous day, looking at the carrying out of the tensile tests for metals, he found himself wondering when the bar would break, and wondering whether the man could say where it would break. Though pieces of metal were taken out of the same mass, the same casting, and were all presumably of the same composition, there was, in each, a spot of greatest weakness, which it was impossible to ascertain beforehand. It was very similar in the case of the human being; a vascular change would affect some part of his brain specially, but which part could not be known beforehand; it would depend to some extent on the individual's constitution, perhaps also on his hereditary predisposition.

He wanted to allude specially to hyperpiesis, because in senile and pre-senile conditions one could apply remedies without using any agent to decrease tension. Where there was hypertension, rest and the proper regulation of the life soon produced a remarkable change, the confusional episodes, coma, etc., disappearing. He had had patients whom he regarded as senile dements, but he found that they had hypertension, and with rest and care they improved enormously, the picture cleared up, and they left the institution. Later, however, they might return several times. In some cases the last time they came back it was found that an element of dementia had crept in, in addition to the confusion and the other episodic conditions; they were now found to be in terminal stages in which there was no doubt about the senility; the picture remained that of a senile dement. Those studies of the circulation could be very helpful to the physician if he could only come to some conclusion as to whether or not the picture was dependent on arterio-sclerosis. The hope he had derived from treatment of the arterio-sclerotic patient was so great that he hoped it would be possible, by further study, to apply it to others. The disconcerting fact was that one often found the picture of arterio-sclerosis without any distinct nervous condition, at least for a long time, and again one often saw arterio-sclerosis without hyperpiesis, and those were difficult cases. There were many cases of real arterio-sclerosis in which the vessels were not atheromatous, and in whom there had been no hypertension during life.

Much more could be said in connection with this subject, but he was anxious to hear others who would contribute. On the previous day he had referred to the lack of co-ordination between the pathological findings and the clinical symptoms, and that was very striking. It could perhaps be accounted for by predisposition, and perhaps by accident in the course of the disease.

Prof. D. SLIGHT congratulated the readers on their papers. He said that a superficial impression of the last two days' debate might lead one to believe that the whole answer to the question of the causation of mental disorder was to be found in abnormalities in the brain circulation; but he thought the speakers had been careful to state reservations and limitations. Dr. Meyer did so on the previous day, and Dr. Guttmann had done so very definitely to-day. Dr. Guttmann had said that some part of what he was saying with regard to circulatory changes in the brain was based on a consideration of the peripheral changes, i.e., there was a presumption that because certain changes were obvious in the periphery, similar changes were presumably taking place in the brain. He, the speaker, raised this question in reference to the mental symptoms found in association with hypertension: was one justified in regarding mental symptoms as due to the circulatory changes in the brain, or must not one consider the possibility that mental

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phenomena and vascular changes were part and parcel of the same process of nervous discharge? Migraine was mentioned. Text-books of medicine had taken it for granted that migraine was based on vasomotor change in the brain. That there might be such a change in the brain was possible, but as one studied the variations of migraine one became more and more convinced of the possibility that any vasomotor changes which took place in the brain were merely part and parcel of a general nervous discharge. That was most evident when, under treatment, one saw a person with migraine change to a patient with obsessional neurosis, or, without treatment, pass over to depression. These phenomena were noted many years ago.

Therefore Dr. MacDonald had raised a point which he, the speaker, also wished to bring up: that psychiatrists might be over-emphasizing the importance of the circulation if it were regarded as the primary factor in the production of some of these mental symptoms—he was referring to the functional circulatory disorders of the brain. He did not know whether the speakers had been unduly fearful of raising psychological questions, and felt under an obligation to speak in gross organic terms. Nothing had been heard of emotional hypertension. He was using the word "functional" in the broad sense, and he would have liked to have heard some statement as to the possibility of vasomotor changes in the brain being caused by truly psychological factors.

Again he congratulated the speakers, and commended them on the fact that two of the papers included many skilful and careful reservations, though they "soft-pedalled" those reservations. Personally, he wished the readers had been more frank about those reservations, so that listeners might have been given a better perspective, as in picking out one aspect and hammering at it for all it was worth there was the danger that one might lose the general perspective.

Dr. F. A. PICKWORTH first wished to congratulate the speakers because their work opened up a new insight into the ætiology of mental disorder, namely, through the vascular pathology which he himself had tried to explain on the previous day.

Dr. Krapf had said that one could compare systemic vascular pathology with that which occurred in the brain; but Dr. Guttmann said that one could not compare the vascular pathology of the systemic circulation with that of the brain. There were certain anatomical differences between the circulation of the brain and that of the systemic organs; e.g., the nerve-supply of the venules to the pericytes of the systemic circulation was absent in brain capillaries.

As to the occurrence of swelling, that might be due to acidic swelling of colloid. Very slight ischæmic changes caused acidic changes in nerve tissue.

The question had been asked whether a lesion did not need to be very extensive in order to produce mental disorder. As a fact the changes might be very small, especially if they occurred in certain regions of the brain-stem and disturbed sympathetic-parasympathetic function.

With regard to the question whether these vascular changes were dependent on nerve stimuli; nerve action was a very rapid phenomenon, but it could undoubtedly initiate changes of capillary patterns; it was, however, not the only cause of variation of such patterns, since metabolic products, circulating hormones and local chemical action also acted independently.

The PRESIDENT thought that in the descriptions to-day speakers were confusing physiological function with mental function. He saw the need for a Glossary Committee of the Association, so that it would be clear what was the meaning of every word used. Other countries might perhaps be persuaded to do the same. Until that was done, progress would be retarded. Reference was made to vascular changes, to blood-vessel walls, and in dealing with circulatory changes blood-flow was mentioned, without saying whether rate or amount of flow was meant. The composition of the blood, too, could not be left out of account. No particular physical defect would cause characteristic mental symptoms.

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Dr. KRAPF, in reply, apologized if he had unduly " soft-pedalled " his remarks ; he was anxious not to go beyond the things which he knew, or thought he knew.

It was not necessary to say very much in reply to the first question, as to what was to be understood by functional circulatory disorders. Dr. MacDonald had already stated the case. In fact one spoke of functional circulatory disorder, because pathological histology had shown that the same histological changes in the brain as were found in cases of vascular disease could also be found without any definite anatomical vascular findings. Evidently there was no other explanation for those histological findings than the assumption of there being functional circulatory disorders at work, i. e., disorders of the actual consecutive function of the brain vessel. It might be said that if there was a toxic product at work in setting up that circulatory disorder, that toxic agent might be the actual cause of the disturbance. Nevertheless it was known that the same circulatory disorders could be brought about not only by chemical, but also by mechanical and nerve stimuli.

Therefore he thought there was justification, in the present state of knowledge, in speaking of " functional circulatory disorders ".

The question had been asked how it could be explained that arterio-sclerosis was such a constant finding in brain vessels, and nevertheless that arterio-sclerotic mental disturbance was relatively rare. In that connection he reminded the meeting that Bergmann said that arterio-sclerosis would be regarded, in the future, as much more an anatomical disease than a clinical one. Arterio-sclerosis was found in cases, but that finding did not tell one anything about the function of the arterio-sclerotic vessels. The question of arterio-sclerosis was also the central point of Dr. McCowan's remarks. His, the speaker's, idea in separating arterio-sclerosis from hypertension, in relation to their mental effects, was two-fold. First, he tried to single out from the symptom of hypertension what clinical pictures could be observed there. Then he made a control by taking 34 cases which were found, at the autopsy, to have very marked arterio-sclerotic changes, but which nevertheless, in their clinical history, showed no evidence of the existence of hypertension. In the pure cases of arterio-sclerosis, which Dr. MacDonald referred to, he had found a marked difference. The clinical picture was characterized by a gradual slow deterioration and dementia, very much in contrast to the rich symptomatology of hypertensive psychoses.

Dr. McCowan had asked if there was a connection between rises in the blood-pressure readings and the occurrence of twilight states. On that point he, Dr. Krapf, could not have expressed himself clearly, because he did not believe that twilight states always occurred after a rise in the blood-pressure. If one took the case of a patient with high blood-pressure, and took the readings every five minutes, in an hour's time in all labile cases there was a constant up and down pressure, and not every variation was accompanied by mental change. He thought that the labile hypertensive patient had his vessels in a constant state of irritation, and that when special conditions came into play this lability expressed itself by functional and vasomotor disturbances in the brain vessels, and those led to the mental disturbances. It was not his view that a lowering of the blood-pressure in those patients should be the object to be aimed at in treatment, because lowering of the blood-pressure was as much a symptom of lability as was the rise in that pressure. What one should strive for was to get the blood-pressure stable, even though that stability was at a high level. It did not matter if the pressure was 200 mm. so long as it was not going up and down and so putting the vessels of the brain under strain.

He did not find himself able to answer all the questions which had been asked in the discussion, but he would like to comment on one remark made by Dr. MacDonald, namely, that we were still in the stage of speaking of mechanisms, and not of pathology. He, Dr. Krapf, agreed, but as those working at the subject were not yet able to go very far in the matter of ætiology, it was necessary to satisfy oneself for the present and be content to study at least the mechanisms ;

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and it might be that by further study of mechanisms the ideal in psychiatry might be arrived at, which would consist of a real attempt to make a pathogenic structural analysis of the mental patient.

Dr. GUTTMANN, in reply, agreed with the remark of Dr. Krapf concerning the last link. He also agreed with the President's remark concerning nomenclature. He would like to explain that he never used the term " functional " as synonymous with hysterical, but rather as opposed to structural. He agreed that the general nervous discharge might be behind these mechanisms which had been discussed. He regarded peripheral vascular changes as indicators of what was happening in the vascular system.

Referring to Dr. Petrie's remarks, he, Dr. Guttmann, was afraid there was no time to enter into the mechanisms involved in the knock-out. He agreed with what the President said about anxiety. He had tried to make clear that it was necessary to analyse types of anxiety states. There were many mechanisms which belonged to this clinical entity, and only after the clinical and psychological aspects had been analysed would it be practicable to speak of the vascular mechanisms underlying them.

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Clinical Notes.

A FATAL CASE OF *STATUS EPILEPTICUS*, WITH POST MORTEM FINDINGS.*

By A. M. WYLLIE, M.B., Ch.B., B.Sc.,
Clinical Pathologist, Crichton Royal, Dumfries.

MISS H. R. N—. Date of birth, November 11, 1889.

She had scarlet fever at the age of 6 years.

She was quite normal till 25 years of age, when she had to give up nursing on account of physical disability, and then suffered from mental instability and confusion for three months. She kept well for eight years.

In 1922, when aged 33 years, she had her first *petit mal* attack, then, one year later, *grand mal*.

In 1925 she showed mental impairment with attacks of confusion. She had fits once a fortnight, treated with luminal gr. j daily. Luminal was found to suit her better than bromide. She manifested the typical epileptic temperament, and was worse in the autumn, becoming elated and grandiose, thinking that the medical officer was the Prince of Wales, that her thoughts were being read, etc.

On August 10, 1936, she commenced having daily fits. Luminal was increased to 4 gr. daily. On August 15 she entered *status epilepticus* at 9.25 p.m. From this time till 7 a.m. on August 16 she had fifty-one major fits. The convulsions started in the right hand and arm, and spread over the whole body. She was treated with chloroform and luminal gr. iij subcutaneously. Morphine was tried but seemed harmful. Lumbar puncture produced no improvement.

On August 17 she passed into a deep coma, the fits having ceased. Temperature was 100·4° F., pulse 130 per minute, respirations 48 per minute. Knee-jerk was absent, feeble Babinski; pupils reacted to light. Her strength gradually failed during the day.

POST-MORTEM FINDINGS.

The post-mortem examination was carried out 5½ hours after death. Post-mortem lividity was present and post-mortem rigidity marked. The pupils were equal. Permission was given to examine only the head.

The dura mater was dense and firmly adherent all over the cranium. The

* Read at the Annual Meeting of the Royal Medico-Psychological Association at Ayr, July 9, 1937.

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brain on removal was found to weigh 50 oz. The convolutions were smoothed out, and there was increased consistence of the brain substance—a condition of brain swelling. Small surface defects were found near the tip of the temporal lobe and in the inferior temporal gyrus, the so-called "*état vermoulu*" (Pierre Marie). The arteries at the base of the brain appeared normal.

Microscopic sections of various parts of the brain were prepared by Nissl's method, Cajal's gold sublimate impregnation, and hæmalum and eosin.

In the *post-central convolution* of the left side a few microscopic lesions were discovered. Stained by Nissl there appeared, in a streak, a condensation of microglial cells. The edges of this streak were not sharply delineated, and merged into the surrounding brain-tissue. In the gold sublimate section the streak appeared as a pale, unimpregnated area, separated from the normal brain-tissue by a zone of proliferated astrocytes. In the hæmalum and eosin section the streak appeared as a very vascular area, densely populated by microglial cells. Imperfect mitotic figures were seen. No corresponding lesions were found on the right side.

Frontal pole (left and right): Very well supplied with blood-vessels. Tangential layer was broad and well delimited. The layers were distinct and well-populated with nicely-stained cells. There was no lipochrome degeneration of the cells. The nuclei were centrally placed and contained a central nucleolus. Round some of the larger vessels of both grey and white matter there was an exudate of round-cells in the perivascular lymphatics. Astrocytes were numerous and well shown in the white matter, and could also be seen in the grey matter.

Precentral convolution (left and right): There was a conspicuous absence of Nissl granules in the cells of both sides, though otherwise the cells appeared healthy. Very few astrocytes could be seen in the left side, but were more numerous in the right. There were patches of closely-packed corpora amylacea in the tangential layer.

Temporal pole: The cells were seen in regular layers. Astrocytes were abundant in the white matter. There were some corpora amylacea in the tangential layer.

Occipital pole: There was a great abundance of capillary blood-vessels; also in the pia arachnoid. Astrocytes were well seen. The cells appeared numerous, but in their normal distribution.

Basal ganglia: The cells were abundant and well stained. No foci of injury or inflammation could be seen. Astrocytes were well seen in the white matter.

Mid-brain and pons: The arrangement of the cells appeared normal, and the cells were normally pigmented. Astrocytes were not prominent.

Junction of medulla and spinal cord: Corpora amylacea were present in the posterior columns. The central canal was obliterated by proliferation of the lining cells.

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Cerebellum : Showed characteristic arrangement and abundance of Purkinjē cells.

Inferior temporal gyrus : The surface presented a breach of continuity. The erosion had undermined edges. The brain-tissue of the floor was œdematous and degenerate, with loss of nerve-cells and fibres. There were some tiny hæmorrhages.

Three findings are of interest :

1. Brain swelling, probably associated with the toxic state in which death occurred.

2. Surface defects ; *état vermoulu* of Pierre Marie ; no doubt traumatic in origin, as the patient had had many falls.

3. A microscopic proliferative lesion in the left post-central convolution in the area for the common sensation of the arm and hand. The whole of the affected part was approximately 3 c.mm. The most likely explanation seems to be that it is the late result of an ischæmic softening, and it is an interesting coincidence that the convulsions began in the right hand and arm. I do not suggest that this tiny lesion was the cause of the epilepsy, but that it might be the only structural expression of a more widespread functional disorder of the vessels, which may have been the underlying cause of the epilepsy.

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Part II.—Reviews.

Administrative Psychiatry. By W. A. BRYAN, M.D. London: George Allen & Unwin, Ltd. Pp. ix + 349. Price 15s.

There is, indeed, a scarcity of books dealing with the administration of mental hospitals, and although this book is written by an American from the point of view of American psychiatry, so many of their problems are the same as ours that this book cannot fail to be of the greatest use to all medical officers of mental hospitals, young and old. The book is not purely theoretical, but written from a very practical standpoint, and more particularly from the view-point of the Worcester State Hospital, Mass.

We wish the following quotations could be permanently before the eyes of some medical superintendents and many members of committees:

"To stress the breeding of blue-ribbon cattle or the production of prize-winning vegetables and sacrifice medical supplies and equipment is a short-sighted policy."

"The custom of judging the administration of hospitals exclusively on the basis of per capita cost is a vicious practice. They can be operated at such low cost that the essential purpose for which they are built is defeated. Excessively low per capita costs are invariably gained by sacrificing many things that would contribute to the recovery of the patients."

It is very interesting to read that "working on the theory that the institution has a double function of medical practice and business management, a few states have tried the experiment of placing a medical director in charge of the treatment of the patients and a business manager in control of the economic side of the organization. This is an administrative absurdity, since such dual-headed forms of organization violate the major premise of good administration—one responsible head to every activity". We could have told them this thirty years ago, when the same idea was found to be a dismal failure in this country, and it always will be. "In order to carry out the highest psychiatric ideals the medical executive must have financial control." This agrees entirely with our views. Certain bodies in this country are trying the same again without any thought of the welfare of the patients.

Again, "When political considerations enter the hospital, professional efficiency flies out of the window". Recent happenings in the State of Massachusetts make our hair stand on end.

The author wisely points out that loyalty works both ways. One has so often seen loyalty converted into an opinion that utter inefficiency must be winked at and quietly hidden.

The goal of a mental hospital is purely psychiatric, so that it ought to be explained to all administrative individuals that their work has to be integrated and correlated with the end in view of promoting the recovery of the patients and nothing else.

The chapter dealing with the nursing problem is full of good advice. We wonder how many of our nurses can be described as "waste conscious"?

We are very pleased to see the statement that eight hours is a sufficiently

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long period for anyone to spend in a ward with psychotic patients. On the other hand, we do not agree that it is impossible for anyone to work ten or twelve hours under the conditions that prevail in the mental hospital. We see it in practice every day. There are two peak hours for accidents in the wards, between 6–7 a.m. and between 3–5 p.m.—both times when nurses who are tired show irritability and lack of care.

The remarks on barbers' shops and beauty parlours make interesting reading, and might with advantage be applied to a considerable number of mental hospitals in this country. We wonder how many mental hospitals with 2,000 patients in this country have 350 major surgical procedures per annum? We do not agree with the "careful selection" of books for the mental hospital library. There would be very few readable books left if some selectors had their own way. Patients should be allowed to read those books which they would choose to read if they were at home. We like the suggestion that the hospital should send each patient a birthday card on his or her birthday.

We think that medical superintendents cannot but derive great benefit by reading this book, and we hope it will improve their attitude towards the assistant medical officer—which is not always what it should be—and lead them to spend time in instructing those with less experience and knowledge than themselves.

We should like to see a copy of this book presented to every medical officer on joining the staff of a mental hospital with instructions to learn it by heart and apply it—with discretion. It is very good.

G. W. T. H. FLEMING.

Treatment in Psychiatry. By OSCAR DIETHELM, M.D. New York: The Macmillan Company, 1936. Pp. xvi + 476. Price $4.

This is the best book on the treatment of mental illness in the English language. This is no compliment, for there are really no others which cover the whole ground. The author, who is Professor of Psychiatry at Cornell University, is a wholehearted supporter of Adolf Meyer, so that much of the treatment indicated is based on Meyerian principles, and follows the lines used in American State hospitals.

The first six chapters are devoted to the general principles of treatment and the last eleven to details.

Psychotherapeutic methods are well covered, but rather too briefly, only sixty-seven pages being devoted to suggestion and hypnosis, psycho-analysis, individual psychology, analytical psychology, group analysis, Rank's and Stekel's modifications of psycho-analysis, persuasion, re-education, and indirect methods. A separate chapter is then devoted to distributive analysis and synthesis as outlined by A. Meyer. The analysis is "distributed" by the physician along the various lines which are indicated by the patient's complaints and symptoms.

Every analysis should then lead to synthesis, so leading to integration of the personality. It is interesting to read the author's views on prolonged narcosis: "Intravenous somnifen, which was used in the beginning, has been given up because of its dangers. Luminal subcutaneously was first substituted and then dial ciba intramuscularly. The most widely used treatment now is the administration of dial by rectum." We wonder how these views would be received in this country!

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We would have liked to see some mention of the use of acetylcholine and of ephedrine in manic states, particularly from a prophylactic point of view. Insulin treatment is considered to be "by no means ready for therapeutic utilization". This is a strangely conservative point of view to hear from America, but this is not the first time we have heard it.

The account of the various methods of electropyrexia is much too brief to be of any value. The index might, with advantage, have been made fuller.

G. W. T. H. FLEMING.

Research in Dementia Præcox. By NOLAN D. C. LEWIS, M.D. The National Committee for Mental Hygiene, U.S.A. Pp. xi + 320.

The author, in surveying the ground for this book, visited about 200 laboratory, hospital and other scientific centres in America.

The author first gives a chapter on the clinical features of dementia præcox and so clears the ground of a good deal of confusion. He favours the time-honoured division into simple, hebephrenic, catatonic and paranoid types. It is pleasing to read his favourable remarks on psychology. There is a distinct tendency, probably largely among those whose knowledge of modern psychology is very small, to say that psychology is of little practical value. Academic psychology has a great deal to teach psychiatry, and one has only to turn to the estimation of deterioration in a patient to see how hopelessly wide of the mark the older psychiatrists' views are compared with the results as obtained by modern methods of ascertainment.

The writer gives a number of questions relating to deterioration which are in urgent need of investigation on scientific lines. How little the language defects in schizophrenia have been studied! How much work remains to be done on problems of child psychology and the pre-psychotic conditions about which we are almost completely ignorant! When discussing ætiology the writer says quite candidly that we know very little. He raises a number of interesting questions about the occurrence of psychoses in primitive peoples, i.e., at what social level in development do psychoses in general and dementia præcox in particular become manifest. What do we know about the distribution of the various types of dementia præcox amongst the various races, religions, social levels, political creeds, etc.? There is a wide field for ethnology here, requiring many years of hard and difficult work.

The author points out that his own researches support to a considerable extent the views of Mott on the changes in the interstitial cells of the testis. He mentions the variously mooted relationship between tuberculosis and dementia præcox. Recent work in this country has answered this question in the negative. He points out the immensity of the task facing those who would set about investigating the neuro-histology of the areas around the third ventricles by the newer histological methods. It is interesting to read that there were in 1936 at least thirty laboratories in America working on the Berger action currents. How little we know really about the volume of research that is being carried on in the world! What do we know about the male hormone component of the urine in homosexuality? Why is theelin found in unusually large amounts in homosexual males? What of the presence of specific hydrolytic enzymes in the blood of schizophrenics? When we come to study brain chemistry we find ourselves hopelessly ignorant. Much work

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has been done since the early work of Thudicum on phrenosins, etc., but what an amazing amount of work remains to be done! Work has been done in recent years on bromine and iodine content, ammonia and adenylic acid, non-protein nitrogen, amino-acids, colloidal states of lipoids in normal and pathological brains, shifting in the ratio of lipoids, phosphatides, cerebrosides, organic phosphorus, sulphatides, cholesterol, glutathione, lactic acid, and the enzymes, including cytochrome and the dehydrogenases. Work is required on nutritional disturbances, including starvation,the chemical nature of the Nissl bodies, the significance of phosphocreatine, and the effect of acetylcholine and eserine on mental states. Much work is required on subjects like colloids, refraction, osmotic pressure, and the fibrin content of blood-serum.

In his conclusions the writer points out that what is urgently required are "Correlated individual statistics on the sociological, clinical, physiological and biochemical aspects, i.e., many types of studies on the same individual case and on the selected groups. An ideal team would include a sociologist, a psychiatrist, a psycho-analyst, an experimental psychologist, an anthropologist, an internist, a physiologist, a geneticist, a chemist, a pathologist, and an expert librarian of the polygot type, with the broad-visioned, clinically trained psychiatrist to serve as the binding-post, else the investigation would enter a multitude of diverse channels leading away from rather than towards the central object of study. It should be emphasized that co-ordinators must be very carefully selected, as this function cannot be carried out by those with strongly prejudiced minds; moreover, very few men are able to evaluate widespread visions, situations and needs, and at the same time focalize intensively on a central problem. We cannot remember ever hearing the problem of the direction of research so well stated. So much time and money has been and is being wasted on futile research on an obsessional basis and almost completely devoid of vision, and of control by normal material.

There is an extremely well-chosen and wide bibliography at the end of the various chapters. We can recommend this book very strongly to those who wish to gain an idea of the immensity of the problem in dementia præcox. We would warn them that the problem is considerably larger than even this book indicates. G. W. T. H. FLEMING.

Principles and Practice of Recreational Therapy for the Mentally Ill. By J. H. DAVIS, M.A. London: William Heinemann (Medical Books), Ltd. Pp. xviii + 206. Price 15s.

This book gives an account of the practice and principles of resocializing physical therapy. Recreational therapy is defined as "any free, voluntary and expressive activity, motor, sensory or mental, vitalized by the expansive play spirit, sustained by deep-rooted pleasurable attitudes and evoked by wholesome emotional release prescribed by medical authority as an adjuvant to treatment". We do not feel altogether confident that some of the recreational activities given as therapy we have seen applied were "free and voluntary" or accompanied by "pleasurable attitudes".

The book is called the "Principles and Practice", and in the contents Part I is described as the principles, but there does not appear to be any Part II, which we should have presumed to be the practice; however, the practice is duly presented. Chapters are devoted to education and re-education, interest and effort, classification of activities, tests of various kinds, various

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exercises, and finally the aims and objectives of recreational therapy. We wonder whether swimming is altogether a safe exercise for suicidal patients.

The book serves a very useful purpose for those who wish to apply recreations in a systematic manner—given the staff to do it with. Individual attention is very necessary in most cases.

The proof-correcting might have been more carefully done, i.e., "catonic" for "catatonic", and "exogeneous" for "exogenous". We would like to see a different definition of psychotherapy than "treatment of disease by analysis, mental suggestion and persuasion". G. W. T. H. FLEMING.

The Intellectual Functions of the Frontal Lobes. By R. M. BRICKNER, M.D. New York: The Macmillan Company, 1936. Pp. xvi + 354. Price 15*s*.

The author gives an excellent and most interesting account of the clinical results of the surgical removal of the larger part of both frontal lobes.

The symptoms showed only an impairment of the completeness of mental processes, but no alteration of their fundamental nature. The only real function which the author considers as primarily affected is the elaborate association into complex structures of the simpler engrammic products associated in the more posterior parts of the brain. He concludes "that the frontal lobes are not intellectual centres in any sense except, perhaps, a quantitative one, and that they play no specialized role in intellectual function. They add to intellectual intricacy in a quantitative manner only, by increasing the number of possible associations between engrams which have already been aggregated to a complex degree in other parts of the nervous system".

The clinical and experimental work is given in great detail.

G. W. T. H. FLEMING.

The Self in Psychology. By A. H. B. ALLEN. Kegan Paul, Trench, Trübner & Co., Ltd.

The Logical Structure of Science. By A. CORNELIUS BENJAMIN. Kegan Paul, Trench, Trübner & Co., Ltd.

Towards a Law of Creative Thought. By ROSAMOND E. M. HARDING. Kegan Paul, Trench, Trübner & Co., Ltd.

One feels justified, though the author in each case may quite properly be outraged by the idea, in including these three books in a single review. No insult is intended, nor is the reviewer more than usually fatigued. No doubt the authors will realize that sparsity of comment is not necessarily paucity of interest.

The reason for classifying these books together is that all three are preoccupied with those unifying tendencies existing in science to-day. Miss Harding's book deals with that contagion of inspiration whereby the same stimulant motive is reflected in different spheres of activity. Mr. Allen's book, *The Self in Psychology*, is of a nature to be expected at a time which sees the activity of the Marburg School, and Mr. Benjamin's thesis on *The Logical Structure of Science* aims at the elucidation of the inter-relationships between the different component subjects of science.

Miss Harding's book will make no appeal to those readers inoculated with

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the present-day love of matter for its own sake. Much of it is occupied with data of a peculiar nature, varying from reproduction of the weird symbolism of the Belisha beacon to charts annotated "world war". (Possibly the author brilliantly anticipated Mr. Hore-Belisha's translation from the supervision of motor traffic to the less hazardous possibilities of universal carnage.) I feel that the nature of Miss Harding's data, and the relative paucity of her explanatory material, will mitigate against her reputation. The dish is not sufficiently meaty for a generation reared on the varied condiments of psychological nomenclature. From this point of view the trouble with Miss Harding's data is their general availability. They are derived by the use of the ordinary senses, without the intervention of multitudinous diabolic innovations. They will, therefore, attract little attention in an age where the mechanical device is a substitute for the higher mechanism of thought. Again, a preponderance of data, and a minimum of inference, is the best basis for scientific investigation. This state of affairs obtains in Miss Harding's work. Her chapter headings, indicative of the themes outlined in ensuing pages, form an idea which might profitably be extended to much psychological writing. It is, after all, better to be let into the secret first than not at all. I look forward to Miss Harding's next book, which ought to deal with current tendencies common to different arts.

The main weakness of Mr. Benjamin's book is the first chapter. This is a wholly laudable state of affairs, as obviously involving no tendency to flatter to deceive. Yet in discussing human activities it is surely too all-inclusive to say that the aim of religion is reconciliation with Nature. What of the Nietschian doctrine that the aim of religious morality is the corruption of nature? There are, however, few such flaws in the construction of this scholarly thesis. While the academic sources of the author's learning extend with appropriate catholicity well into the past, the main themes of the book are very much in the current mode. He sees clearly the fallibility of that concept of scientific endeavour which discloses the nature of things by a ruthless and continuing mathematical subdivision. He exposes very completely the limitations of the pseudo-rationalism of mathematics. Possibly one's pleasure in this aspect of Mr. Benjamin's work is misplaced, and cannot adequately be held to represent a true scientific opinion. The reviewer, it may be said, has never derived any very considerable pleasure from the hectic frenzies of mathematics, and the transient mutual agreement that two and two make four appears to him to be a no more secure basis for investigation than the pathetic hypotheses of certitude put forward by the League of Nations. The book, to me at any rate, conveys much salutary correction for the worst errors of psychometry.

Mr. Allen's book, on *The Self in Psychology*, is an important thesis. A book dealing with a hypothesis so saliently unifying as that of the self merits at least that first humanity of criticism, of being dealt with as a whole. One can praise no more than by asking that it should be read. It is carefully thoughtful and fair, and understandable without being too obvious. Scientific works too often fall into two main categories, from the point of view of readability. Some are as vacuously clear as the simpler motives which illumine those weeklies, written for domestics, which now hold an exclusive corner in the literature devoted to the mill girl and the humane baronet. Such works have a simplicity less beautifully virtuous than might appear at first sight, being bred by dogma out of limitation. At the other pole we have those finely reverberating tomes which leave one spiritually uplifted and mentally

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bewildered. Mr. Allen's book pertains to neither technique. It has the virtue of being understandable, with care. What appears too soon, too obviously, to consist of fact, is apt later to appear among the mere chimeras of science. Mr. Allen's book merits in its reading something of the care which went to its construction. It is an effort which will repay him who makes it.

A. GUIRDHAM.

Mitogenetic Analysis of the Excitation of the Nervous System. By Prof. A. G. GURWITSCH. Translated by G. and F. ENGEL. Amsterdam: N. V. Noord-Hollandsche Uitgeversmaatschappij, 1937. Pp. 141. Price 11s.

Prof. Gurwitsch here gives a collected account of his researches into the activity of nervous tissue as a producer of mitogenetic rays.

These rays are said to be given off by most living tissues and by some chemical reactions, and are detected, as their name indicates, by their power of inducing mitosis in susceptible organisms. Cultures of yeast are commonly used, and, after irradiation, the amount of growth which occurs is compared with the growth occurring in a control culture. There is, at present, no other method of detecting them. According to Gurwitsch, they may be refracted by a quartz prism and have a wave-length of 1900 to 2500 Angström units.

Unfortunately there is some disagreement as to whether such rays even exist. So far there has been little work done on the subject except in Gurwitsch's laboratory in Leningrad.

The book gives a large amount of information in condensed form, but so few details of practical work are given that it is difficult to form any opinion of the value of the work. On the other hand, it seems to be almost impossible that so many results, and many of them consistent with one another, could be the result simply of random errors and self-deception. He mentions in the preface that the book is based on over 5,000 experiments, and that eleven collaborators and guests took part in the work. Further, he says of several of the crucial experiments that they have been verified repeatedly.

One of his more important conclusions is that there are a number of ways in which nerve tissue may become excited. In classical physiology there is recognized at present only one way, namely, by the passage of an electrical impulse. Gurwitsch, however, exposes nerve-tissue before the slit of a quartz spectroscope and, where the spectrum would fall, places a row of capillary tubes each containing yeast culture. Some of the cultures react and some do not. In this way the spectrum of mitogenetic rays may be explored. He has found that a piece of nerve, when excited by different methods (e.g., mechanical crushing, faradization, etc.), emits a different spectrum for each method. He concludes that there are many ways in which a nerve-cell may become excited. Further, he gives evidence to show that for some of the modes the "all or nothing" law does not apply.

Finally, he suggests that as nerve tissue emits mitogenetic rays when active, and is also affected by the rays when acted on by them, it is possible that mitogenetic rays may play a part in the functioning of the nervous system.

The method needs confirmation by other workers before one can accept it unreservedly, but it is certainly worthy of attention. It seems to be particularly useful as a method for investigating biochemical changes in nerve tissue.

W. R. ASHBY.

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L'Hystérie et L'Évolution Humaine. By M. DIDE, M.D. E. Flammarion. Pp. 250. Price 12 francs.

The author is more concerned with the portrayal of the hysterical character and the place he takes in a modern civilized community than in the description and elucidation of specific symptoms. The hysteric is a person whose mental growth has been arrested at an infantile level; his thinking processes, akin to the primitive, are infralogical, and his judgments emotionally rather than rationally determined. Fundamentally his state is due to affective poverty. In the sexual sphere this is shown in the female hysteric by her inability ever to arrive at its true goal—the maternal sentiment; while in the male there is a failure to sublimate this instinct towards social ends.

The hysteric masks his incapacity to feel by hypermotivity. There is, characteristically, a dissociation between the faculty of expression and the underlying sentiment. Imitation and suggestion, instead of sincere emotional drives, rule his conduct. Hence the hysteric's theatricalism, his tendency to fabrication and mythomania. These same motives, ultimately an expression of his inner dissatisfaction and emotional poverty, will cause him to grasp at any new cult or "ism", with its possibility of a new sensation. Or again, they will lead him to gambling, swindling or alcoholism. In the "crime passionnel" the motives found are not true jealousy, but egoistic vanity and envy.

Kretschmer's views are dismissed in a few short paragraphs. Janet's theory is more sympathetically reviewed. The author differs from Babinski in that he regards pithiatism as one of the most striking manifestations, but not the cause of hysteria. Psycho-analytical theories are considered to be on a level with the mythomania of the hysteric himself, and there is a hint that they may both have the same ultimate source.

Dr. Dide is an acute observer, and his studies of the varied manifestations of the hysterical character make excellent reading. This is a scholarly and erudite work with frequent philosophical digressions. It can be warmly recommended for leisure hours. It is not a text-book.

STANLEY COLEMAN.

The Psychology of Belief. By H. LUNDHOLM, Ph.D. Cambridge University Press and Duke's University Press. Pp. 245. Price 13*s.* 6*d.*

McDougall has stated that the psychology of belief presents three main problems: (1) the problem of the conditions which engender belief; (2) the nature of the state called holding a belief; and (3) the problem of the nature and proper classification of belief as a mode of being conscious. In the present work an attempt is made to explore the psychology of belief in all these three aspects. Lundholm's psychological principles, while closely approximating to those of McDougall, differ in one important aspect. He holds that during evolution adaptive behaviour becomes differentiated into general impulses as well as the special instincts of McDougall's psychology. The former are—primordial curiosity, the impulse to sleep and primordial deference. Primordial curiosity and deference are called into play by all significant physical events and invariably co-operate with the special instincts.

Lundholm's theory on belief-engenderment and the important part played

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by primordial curiosity is most easily summarized by means of a hypothetical example: Suppose an infant coming across a significant physical event, an apple, for the first time. Under the influence of primordial curiosity he will play with it—that is, test it with his various senses. If, in the course of his play, he chances to taste it, the food-seeking instinct will be aroused. On the other hand, if no special instinct is awakened the incident will be forgotten, that is, the apple will not have become a percept—it will have no meaning. But in the former circumstances when the emotion of hunger next occurs, the food-seeking instinct will combine with the impulse of curiosity in an attempt to find an apple. In other words, the object perceived is the object striven for, and the object striven for repeatedly becomes an object of enduring belief.

For Lundholm there are two essential principles responsible for belief-engenderment—the emotional and the rational. The instance given above is an example of the emotional principle. In the formation of the rational principle primordial curiosity undergoes a remarkable evolution, becoming the impulse which urges and accomplishes the relating of things, events and propositions in the most intricate and complex sense. In the hypothetical example of the apple, the emotionally conditioned belief that hunger can be satisfied by the fruit will be subsequently modified by the rational principle. For instance, the child will learn to discriminate between ripe apples on the one hand and green, rotten or maggoty apples on the other; again, he may learn, perhaps, the disadvantages of obtaining his apple from his neighbour's orchard.

The second main part of the treatise deals with beliefs as enduring mental dispositions guiding human conduct. It propounds a hypothesis according to which a belief, as a unit of structure, is a thinking-striving disposition, its conative constituent being deference to a psychological object as the seat of certain known properties. Belief-formation thus represents the investment of an object with deferent impulsive energy; the abandonment of belief the detachment of this energy.

The third section discusses the subtle problem as to whether there are any attitudes of mind in which we apprehend without either believing or doubting the object apprehended. It is thought that there are probably two such states—that of logical contemplation and that of æsthetic contemplation. In both cases the major motive is a rational impulse derived from primordial curiosity. The total conation of the two attitudes, then, would be primordial curiosity supported by deference to the elements of the æsthetic or the logical proposition. It is noted that in the attitudes approximating apprehension without deference, no special conation ever enters. This indirectly leads to the conclusion that special conation, although it does not necessarily determine the reality of things, yet determines their reality-concern.

The two chapters on the formation of false beliefs in the manic-depressive psychosis and schizophrenia lay themselves open to criticism. In the former we are told a state of dissociation takes place, probably due to a toxin—"a poison of the nature of a depressant drug working upon the higher centres of the nervous system". Delusions here are paramountly emotionally induced beliefs, comparable to the beliefs of the child and of the primitive man. As an explanation of the remission we learn that as the toxic influence upon the brain gradually wears itself out, the rational processes re-assert themselves and the delusions become rectified. The credulity of the dreamer and the distortion of the dream-content "that Freud has attempted to explain in a somewhat fantastic vein" are examined along similar lines.

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Schizophrenia, on the other hand, is probably not due to a toxin, but to inborn egotism. The mind of the schizophrenic is not disintegrated, but pervertedly integrated. It is this perverted integration that results in false beliefs emotionally conditioned and impervious to the rational principle.

An important feature, making the whole a really valuable work of reference, is the detailed reviews and elaborate criticisms of the theories held by leading psychologists. Also a wealth of quotations from poetry, drama and fiction throw interesting side-lights upon the subject. Lundholm's treatise can be confidently recommended to anyone who cares to speculate upon the psychological basis of true and false beliefs. It should prove particularly useful to those engaged upon a psychological investigation of delusions.

STANLEY COLEMAN.

Freud and Marx. By R. OSBORN. Victor Gollancz, Ltd. Pp. 285. Price 8*s.* 6*d.*

Osborn presents a comparative study of psycho-analysis and Marxism. He attempts to illustrate the interrelationships between the subjective life of man as described by Freud, and the objective world of economic processes, whose laws of development Marx has investigated. For the psychologist the most interesting portion of the book will be the two chapters dealing with the unconscious mind and dialectical materialism. He brings forward evidence to show that unconscious mental processes are essentially dialectical in character and consequently at variance with the laws of formal logic, which represent the material world as rigid and static. On the other hand, the conscious ego, which reflects reality, gives a picture which is largely undialectical. For the development of this undialectical, static image of reality two main factors are held responsible. The first of these is a conscious process, and may be termed the isolating character of scientific thought. The scientist abstracts certain factors relevant to his inquiry from their environment, and tends to give them an independent and absolute existence. The second is an unconscious one, and proceeds from the clash between reality and id impulses. From this point of view the distortion of the material world by the conscious ego takes on the nature of a defence mechanism. It represents the environment as harsh, rigid and unyielding in order to strengthen the repression of the id.

For those whose main objection to psycho-analytic tenets is that they run counter to formal logic this book provides a strong counter-argument. Where there is so much that is of interest and value it seems a pity that a more detached and scientific presentation could not have been obtained. It is feared that the uncompromising and violent revolutionary sympathies reiterated throughout the book may alienate many readers.

STANLEY COLEMAN.

The Background of Spiritual Healing. By A. GRAHAM IKIN, M.A., M.Sc. George Allen & Unwin, Ltd. Pp. 224. Price 6*s.*

Originally presented as a series of lectures, their project was to convince an audience, practising spiritual healing from the religious approach, that God, besides working through them, is also working through the developments of science, more particularly psychotherapy. To this end, therefore, their subject-matter is largely a résumé of the psycho-analytic theory, or such of it

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as might be more easily digested by such an audience. A subtle distinction is made between moral disease and sin. The former is due to repressed complexes and is beyond the direct control of the will; it is the province of the psychotherapist. The latter is due to the acceptance of a low ideal, or the deliberate refusal to admit the obligation implicit in a higher one; it is the province of the confessor. There are many mixed cases, and here confessor and therapist should work hand in hand. Finally, in moral disease, after the repressed complexes have been brought to consciousness, the confessor can frequently help the patient, and prevent a relapse by redirecting his interests towards higher ideals. STANLEY COLEMAN.

The Unknown Murderer. By THEODOR REIK. International Psycho-Analytical Library, No. 27. London: The Hogarth Press, and the Institute of Psycho-Analysis. Pp. 260. Price 12*s.* 6*d.*

Interest in criminology, both in fact and in fiction, is already very great and is still growing, and so there is an obvious call for books dealing with the psychology of murder. It may be stated at once that this book does nothing to satisfy such a call. No doubt it gave satisfaction in the writing, but it is hard to see why it should have been translated, unless to fill a gap in the International Psycho-analytical Library. The translation is workmanlike, but uninspired. A sentence such as the following, "Our interest (in crime) would be understandable . . . if it concerned psychical happenings as they appear to the neurologist who is called in as an expert", seems a trifle obscure. Of course it might happen that a neurologist was also an expert on psychical phenomena. Again we meet the inevitable "bad conscience" of translators instead of the more English "guilty conscience". When reading this book one has the impression that the author is laboriously working up to some triumphant conclusion, at which, however, he unfortunately never arrives. Nevertheless he is not deterred from remarking, "Now that I have proved my theory". But what is it? A large amount of space is devoted to proving that circumstantial evidence can lead to false conclusions, and that miscarriages of justice may occur. This, surely, is already too well known to need proof. His psychological explanations of judicial errors are unconvincing and forced: the sadism, the hunting instinct, and the narcissistic pride of the prosecution. More interesting is the idea that psychological reality may be mistaken for material reality—that is, thoughts may be treated as facts. He considers that psycho-analysis has, and will have, no place in the court of law, but will certainly lead to changes in penology and the administration of justice. This may well be, but in spite of the growth of psycho-analysis the administration of justice in certain parts of the world appears to be changing for the worse, and radical reform has taken place in the past, long before the birth of psycho-analysis. He compares the psychopathology of judgments, based on cases of miscarriages of justice, with that of psycho-neuroses: the comparison is very laboured. It is well known that primitive peoples have ideas about murder and the proof of guilt which differ materially from ours, and these facts do not need to be proved by the wealth of anthropological detail which they are accorded here. Probably the average reader's credulity will be strained most by the psychological processes attributed to the detective in the Breese case. For the rest, there is a good bibliography which might be of value to authors of detective fiction. E. N. BUTLER.

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Part III.—Epitome of Current Literature.*

1. Anatomy and Physiology.

The Transmission of Impulses through a Sympathetic Ganglion. (*Amer. Journ. Physiol., vol. cxix, pp.* 221–35, 1937.) *Cannon, W. B., and Rosenblueth, A.*

Evidence is presented as to relative doses of acetylcholine, which produces stimulant and depressant effects on the superior cervical ganglion of the rat. Prostigmine may have a depressant action on the ganglion, but it may potentiate the responses to maximal single shocks applied to the preganglionic fibres. It has no direct action on the post-ganglionic fibres or nictitating membrane. Given shortly before tetanic stimulation of preganglionic fibres prostigmine cuts short the contraction and turns it into a relaxation. The depressant action gradually disappears. The relaxation is attributed to excess of acetylcholine. The bearing of these observations on the chemical theory of synaptic transmission is discussed.

E. D. WALTER (Chem. Abstr.).

Further Studies on the Effect of Lesions of the Hypothalamus on Carbohydrate Metabolism. (*Trans. Amer. Neurol. Assoc., vol. lxii, pp.* 49–52, 1936.) *Cleveland, D., and Davis, L.*

Cushing's observation that stimulation of the superior cervical ganglion in cats evokes hyperglycæmia and glycosuria has been confirmed. This effect was abolished by hypothalamic lesions, by section of the splanchnic nerve or by subcutaneous injection of 1·5 mgrm. ergotamine. The sensitivity to insulin was increased by the production of lesions in the hypothalamus destroying the filiform nuclei or by bilateral removal of the cervical sympathetic trunk and superior cervical ganglion. Fatal hyperglycæmia following removal of the pancreas was prevented by the production of hypothalamic lesions, but not by section of the spinal cord between the fifth and sixth cervical segments. The injection of cerebro-spinal fluid from a diabetic monkey into the cisterna magna of a non-diabetic pancreatectomized monkey with a hypothalamic lesion caused no significant elevation of the blood-sugar.

MARION HORN (Chem. Abstr.).

The Nervous Regulation of Pituitary Function. The Consequences of Physio-pathology and Therapy. (*Presse Méd., vol. xliv, pp.* 1521–3, 1936.) *Roussy, G., and Mosinger, M.*

Neuro-regulator centres of the hypophysis are located in the hypothalamus and in the superior cervical ganglion, which itself is under control of the hypothalamus. The centres in turn are under partial influence of the pituitary hormones. Extirpation of the superior cervical ganglion causes in the dog pituitary hypersecretion, with resultant histo-physiological reactions in the vegetative neurons of the hypothalamus.

A. E. MEYER (Chem. Abstr.).

* A number of abstracts in this section are reproduced from *Chemical Abstracts* by kind permission of Prof. Crane, of Ohio University, to whom the Editors wish to express their thanks.

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Pathological Changes of Peripheral Nervous System under the Action of Electrical Currents. (*Sov. Psychonevr., vol. xii, No. 6, p. 5, 1936.*) *Zolotova, N. A., and Zinkin, A. M.*

The present work is a continuation of a series of systematic researches on electrical trauma in acute experiments and in those of long duration on animals (during three weeks after the first electrical trauma). This paper deals with the results obtained on peripheral nerves and muscles.

The authors experimented on dogs, using direct and alternate currents of a strictly known intensity and power; the resistance of the body of the experimental animals was also measured. The voltage used was 120–200 and 450 v.; milliamperage, 65 m.a., 130 m.a., 160 m.a., 350 m.a. The time varied between 12 seconds and 56 minutes in acute experiments; the current was applied either once or many times in different tests.

For control tests with the animals surviving, the current of 90 m.a. and 120 v. was applied during 9 minutes.

The electrodes were applied either to the head and to one of the extremities or to the extremities alone.

The pathological changes in muscles (not in the immediate vicinity of the electrodes) consisted in œdema of the tissue, disappearance of transverse striation and swelling of cell nuclei.

In the vicinity of the electrodes the muscle tissue was of homogeneous aspect, weakly coloured. On the background of comparatively intact fibres there were strips of structureless tissue, sometimes disintegrating into separate clots.

In the nerves the changes were different in parts close to or remote from the application point of the electrodes. In remote parts there was œdema, swelling of tissue, hyperæmia and sometimes small hæmorrhages, all these phenomena increasing in their intensity towards the electrodes and the hæmorrhages becoming greater.

At the actual place of application of the electrodes the changes were of a different nature; there was disintegration of nerve-tissue with fusion of myelin sheaths and axons, and sometimes fusing together of the nerve with the adjacent muscles.

The authors attempt to foresee the processes that would take place if the animal remained alive; the possibility of the restitution of function when the changes consisted in œdema with small hæmorrhages; cicatrization in presence of large extravasations, destruction of the nerve and its fusing together with underlying muscles. (Authors' Abstr.).

Role of the Central Nervous System and Thyroid Gland in the Heat Regulation of the Organism. (*Ber. Ungar. Pharm. Ges., vol. xiii, pp. 432–8, 1937.*) *Issekutz, B., jun.*

Dissection of the spinal marrow at the neck caused only a temporary disturbance in the chemical heat-regulation system of dogs. After removal of the thyroid glands there was a decrease in the chemical heat regulation parallel to a diminishing of the basal metabolism. Lack of thyroid gland could temporarily be replaced by injections of thyroxine. After dissection of all nerves connecting the heat-regulating centres with the periphery the chemical heat regulation of dogs totally ceased. Thus the existence of a separate peripheral heat-regulating system is most improbable. S. S. DE FINALY (Chem. Abstr.).

Variations in Weight, Water Content and Potassium of the Nervous System from Birth to Maturity. (*Bull. Soc. Chim. Biol., vol. xix, pp. 664–70, 1937.*) *Leulier, A., and Bernard, A.*

Determinations were made on the brain, medulla and sciatic nerves of young rabbits, cats and guinea-pigs of different ages. In all cases the water content decreased with age. In rabbits and cats, which are born almost helpless, the

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potassium of all the nervous tissues gradually increased during the first few weeks of life. In guinea-pigs, which are as active as adults when born, the potassium content changed little if at all after birth. L. E. GILSON (Chem. Abstr.).

Is the Action of Vasodilator Nerves Due to a Liberation of Acetylcholine? (*Compt. Rend., vol. cciv, pp.* 1586–8, 1937.) *Tournade, A., and Chevillot, M.*

The vascular phenomena which follow excitation of vasomotor nerves are not in all cases identical with those produced by minimum doses of adrenaline or acetylcholine. This was found to be the case in an experiment using a dog kidney perfused with blood from the cervical veins of a second dog. It remains to be proved that nervous excitation acts exclusively through an intermediate chemical reaction. D. S. SEARLE (Chem. Abstr.).

The Liberation of Adrenaline and Sympathin Induced by Stimulation of the Hypothalamus. (*Amer. Journ. Physiol., vol. cxix, pp.* 615–22, 1937.) *Magoun, H. W., Ranson, S. W., and Hetherington, A.*

Using the denervated nictitating membrane of the anæsthetized cat as an indicator, the liberation of circulating adrenaline and sympathin by stimulation of the hypothalamus was demonstrated. E. D. WALTER (Chem. Abstr.).

The Dynamics of Vegetative Reflexes under Conditions of Low and High Temperatures and of Ray Energy. (*Sov. Psychonevr., vol. xii, No.* 6, *p.* 16, 1936.) *Liderman, M. J.*

The author studied experimentally the vegetative reflexes in persons who were placed in special rooms with low or high temperatures and ray energy, and came to the following conclusions:

(1) The immediate effect of the low and high temperatures and the ray energy upon man produces certain dynamic changes of the vegetative reflexes according to a well-defined principle.

(2) As a result of long and frequently repeated influence of the temperature upon the human organism, the author noticed a certain improvement of vascular, sweating and thermal reactions.

(3) This improvement was of a double kind: (i) The initial state of vegetative reflexes was already changed before the beginning of the experiment in comparison with the first days of observation. (ii) The development of vegetative reactions in the time of the experiment differed also from that of the first days of observation.

(4) The observed opposite tendencies in the development of vegetative reflexes in two persons, who were under experimental observation, can be explained by the individual and constitutional peculiarities of their organisms (by way of working hypothesis).

(5) The improvements of vegetative reflexes observed by the author show that there is a change in the organism; certain new tendencies are created in response to one or other temperature, which are more favourable for the carrying on of physiological processes. (Author's Abstr.).

Anencephalons and Their Behaviour. (*Sov. Psychonevr., vol. xii, No.* 6, *p.* 32, 1936.) *Sokolanski, G. G.*

The writer gives a detailed clinical and histo-pathological description of a case of anencephalia. The human being that was born full-time lived only about 24 hours. It showed spontaneous movements of the head and extremities, motor reactions to tactile and painful stimuli, combined symmetrical crossed reactions, i.e., in applying painful stimuli to one extremity the contra-lateral one reacted with the same movements as that to which the stimulus was applied. As no brain tissue was found on post-mortem examination, the whole nervous system having been reduced to the presence of insufficiently developed spinal cord, medulla

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oblongata and lower part of pons Varolii (up to the origin of the fifth pair), all the above-mentioned movements must be considered as a manifestation of the reflex activity of the spinal cord. Primitive expressive movements of the face, in absence of the 'tween- and the mid-brain, denote the fact that elementary facial expression was possibly realized by the medulla oblongata and a part of the pons. Clinically no deep tendon reflexes could be revealed that may have been due to pathological changes in the cells of the anterior horns of the cervical and lumbar enlargements of the spinal cord.

Various reflex reactions obtained from the skin (abdominal reflexes, Babinski reflex with "signe d'eventail", spinal reflexes of Veraguth, palmar reflexes including that of Robinson, etc.) were also accomplished through the spinal cord or the brain-stem. (Author's Abstr.).

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2. Psychology and Psychopathology.

The Psychobiology of Anxiety. (*Psychoanal. Rev. Suppl., vols. xxiii and xxiv, July and Oct.*, 1936; *Jan., April and July*, 1937.) *Yuskin, J. C.*

Anxiety is defined as a form of affectivity recognized introspectively as an unpleasant affect accompanied by fear without any known cause, or the cause inadequately understood or mistaken, and manifested objectively by changes in the neuro-muscular, autonomic visceral and secretory functions (emotional expression).

Like other forms of affectivity, anxiety is derived from instinctive tendencies and is related to both conscious and unconscious processes. It is regarded as having its centre in the diencephalon, and may therefore be influenced by disturbances of the various end-organs, by the general chemism of the organism and by cerebral impulses via the cortico-diencephalic pathways.

Clinically anxiety arises in (1) disturbances of the various viscera, organic diseases of the brain, toxic conditions and disturbances of sexual function; (2) situational difficulties when the causes are more or less conscious, and traceable chiefly to conflicts between the ego and herd instincts; (3) conditions in which the causes are unconscious and related chiefly, though not exclusively, to the sex instinct. This section of the monograph includes detailed observations upon twenty-five illustrative cases.

Clinically anxiety may be the outstanding symptom; it may give rise to many accompanying symptoms; or it may be replaced or avoided by the formation of symptoms free of overt anxiety. The severity of the anxiety and of the associated symptoms depends upon constitutional factors, the precipitating causes, the degree of regression and the mode of symptom formation.

Anxiety and its equivalents, together with the various mechanisms developed to escape anxiety, constitute important factors in the psycho-pathology of abnormal personalities, neuroses, psychoneuroses, psychoses, as well as in somatic and toxic diseases.

An evaluation of the causes and the mechanisms in the production of anxiety and its associated symptoms or of the dynamisms which replace or avoid it aids in clinical diagnosis, in prognosis and at times in treatment.

STANLEY M. COLEMAN.

Symposium on the Theory of the Therapeutic Results of Psycho-analysis. (*Internat. Journ. Psychoanal., vol. xviii, p. 125, April–July*, 1937.)

E. Glover sketches the more recent developments of psycho-analytic theory regarding ego-psychology and the effects of introjection mechanisms upon the transference situation. He suggests that the emphasis placed on early phases of introjection and projection has led to a neglect of the fundamental importance of repression, particularly in the later infantile years. He also thinks that analysts

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as they have come to know more of mental development and have become more ambitious in their therapeutic aims, have tended not only to be more obscurantist in their therapeutic ideology, but to over-estimate the refinement of their labour.

O. Fenichel discusses the various means the analyst can use to influence the ego to desist from or to modify its pathogenic defences against the id.

J. Strachey finds that the immediate determinants of the therapeutic results of psycho-analysis are to be found in the procedures of interpretation, and more particularly of transference-interpretation. He also believes that it will only be possible to understand the results of these procedures, and how these procedures are put into operation, if sufficient attention is paid to the mechanisms of projection and introjection.

E. Bergler deals with some of the methods by means of which the ego may be freed from anxiety.

H. Nanberg investigates the transference-resistance in its relation to the repetition compulsion. He finds that in the transference the ego libidinizes the repetition compulsion, and unites with it to the advantage of the treatment.

For E. Bibring the aim of therapy is described as a change in the reciprocal relations between the various institutions of the mind. In the three sections of his paper the basic principles of cure are dealt with from the point of view of the id, the super-ego and ego respectively. STANLEY M. COLEMAN.

Some Anthropological Perspectives on Psychoanalysis. (*Psychoanal Rev., vol. xxiv, p.* 246, *July*, 1937.) *du Bois, Cora.*

Psychoanalysts, in dealing with cultural data, profess to see in social institutions parallels to the developmental sequences they have established for the individual. The writer points out the many pitfalls of the evolutionary theory of culture, which is merely an outgrowth of biological analogy.

An alternative hypothesis of the comparative approach with its single, although basic, assumption of the psychic unity of mankind is put forward. In favour of this hypothesis the following points are made: (1) It is the intuitive impression of most field workers that aborigines do not differ from ourselves in their mental processes; (2) aborigines brought up among western Europeans have adapted themselves successfully to their culture; (3) it has been impossible to discover faculty differences between aboriginal and civilized man, which cannot be attributed to either cultural conditioning or the nature of the test devised; (4) it has been shown that there are certain constants in the dream forms of widely separated peoples living in very different cultures; (5) there are certain large categories of institutionalized behaviour (marriage, incest tabus, religious belief, etc.), which, however different they may be in their forms, indicate a certain unanimity in man's psychic constitution, at the same time that their variety indicates the improbability of a single historic origin.

A possible theory concerning the major components of the human psyche is then put forward, and various fields of inquiry mutually profitable for the anthropologist and the psychologist are suggested. STANLEY M. COLEMAN.

On Introjection. (*Internat. Journ. Pychoanal., vol. xviii, p.* 269, *April–July*, 1937.) *Fuchs, S. H.*

An exhaustive and critical survey of the literature concerning the mechanisms of introjection, projection and identification shows that there is some confusion and looseness of nomenclature. In order to clarify the subject the writer proposes several alterations in the terminology at present adopted. He suggests that analysts should drop the term "primary identification", accepting the infant's view-point of a primary unity of ego and non-ego. To express the child's gradual differentiation between ego and environment he would like to use some special terms, such as "internalization" and "exteriorization". Introjection could then be confined to the "act" of inclusion into the ego complex by way of instinctive pattern—

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phylogenetically speaking an oral incorporation—and identification could be reserved for the " fact " of inclusion into the ego system.

STANLEY M. COLEMAN.

The Relationship between Hysteria and Schizophrenia [*Rapports de l'hystérie avec la schizophrénie*]. (*Ann. Méd. Psych., vol. xcv* [*ii*], *p.* 1, *June*, 1937.) *Claude, H.*

Besides having many clinical features in common, particularly cataleptic phenomena, hysteria and schizophrenia are both considered to result ultimately from the same psycho-pathological process producing a state of dissociation. Whereas in hysteria the dissociation is superficial and temporary, is determined by some emotional trauma and can be removed by persuasion, in schizophrenia it is deeper, more rigid and durable. For these reasons the writer would like to classify hysteria among his schizoses, a group of border-line states, which he places between the neuroses and psychoses proper. STANLEY M. COLEMAN.

An Inquiry into the Variation of Precision of Movement with Age during Adolescence. (*Brit. Journ. Psychol., vol. xxvi, p.* 441, *April*, 1936.) *Sparrow, W. J.*

The writer investigated 300 secondary school-boys of ages 11–16. The test consisted in placing a fine stylus into three small holes in succession. The ability to make the movement changes little from 11–14 years, but increased sharply at 14–15 years, after which there was a further period when no further increase took place. The standard deviations for the right hand are greater than those for the left in the case of the large hole, while the reverse is true for the other two holes. This may be due to either a settling-down process, consisting of an adaptation to imposed conditions and the gradual disappearance of a feeling of the novelty of the task, or to the under-estimation of the difficulty of the task.

G. W. T. H. FLEMING.

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3. Psychiatry.

Analysis of a Case of Schizophasia. (*Acta Psychiat. et Neur., vol. xi, p.* 585, 1936.) *Sjöden, B.*

The patient's language disturbance can be divided into two components—first, disturbances in the train of association, and second, his distorted way of expressing himself by the use of foreign words and neologisms. The best attitude to take towards schizophasia is to regard it from a tripartite angle, combining Kleist's lesional aphasia (a form of sensory aphasia) with Schneider's disturbance in trains of association (similar to the thinking during mental exhaustion), and Gruhle's idea that there is a will factor which helps to create the catatonic features. The addition of " learned " words and expressions is to compensate for the feeling of inferiority. G. W. T. H. FLEMING.

Cryptorchidism Associated with Behaviour Problems. (*Journ. Nerv. and Ment. Dis., vol. lxxxv, p.* 51, *Jan.*, 1937.) *Molitch, M.*

Eleven boys with undescended testicles were treated with anterior pituitary-like hormone obtained from the urine of pregnant women. In 7 the testes descended, and in 3 of the 4 failures there was a mechanical obstruction. All the boys were unstable, emotional, and presented serious school problems. In intelligence but not in performance tests the boys were about the average of the population.

No more than 250 rat units should be given for the first few doses.

G. W. T. H. FLEMING.

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On the Interrelations of Schizophrenia and Heterogenic Organic Diseases of the Central Nervous System. (*Sov. Psychonevr., vol. xii, No.* 6, *p.* 49, 1936.) *Grinberg, M. D.*

The author had under observation 4 cases of a combination of schizophrenia with general paralysis, 3 with syphilis of the brain, 4 with epidemic encephalitis, 5 with trauma of the head and 3 with arteriosclerosis of the brain.

Of particular interest are those cases in which a schizophrenic picture preceded the heterogenic organic disease (such cases were met with in each group).

Based on the assumption that in the origin of schizophrenia both endogenous (hereditary-constitutional) and exogenous factors play a constant part, the author distinguishes between a form of schizophrenia that is allied to organic psychoses, in which the hereditary factors play no considerable role, and the idiopathic schizophrenia in which the chief picture is a schizophrenic one, even in the presence of manifested heterogenic psychoses, and the very development of disease is achieved according to a schizophrenic type. In such cases a heterogenic organic psychosis (for instance, general paralysis) "unifies" the schizophrenic symptomatology. On the whole, both the course and the clinical picture of disease depend on the particular combination of heterogenic-organic and of constitutional schizophrenic agents, and change even in one and the same patient in different stages of the disease. The author is of the opinion that cases of combination of schizophrenia with well-studied organic diseases demonstrate the importance of exogenic factors in the origin and development of schizophrenia and their interaction with the hereditary factors. (Author's Abstr.).

On Certain Hormonal Disorders in Schizophrenia. (*Sov. Psychonevr., vol. xii, No.* 6, *p.* 80, 1936.) *Turkevitch, O. M., and Gershanovitch, Z. S.*

The authors investigated in twenty-one women patients suffering from schizophrenia the content of prolan in the urine and blood and, in some instances, in the spinal fluid. The results were negative. Folliculin was found only in two out of seven cases investigated. Supposing that the absence of hormones in the fluids of the body may depend on the insufficiency of the method itself, the authors investigated also in a series of experiments the hormones in cases of schizophrenia complicated by pregnancy, climate, brain tumour, i.e., by those conditions where the amount of prolan is increased according to the data from the literature; they found that although the content of prolan in such cases was as a matter of fact increased, yet this increase was not of the same degree as in mentally normal persons suffering from the same conditions. This fact denotes that in schizophrenia the prolan content (of urine and blood) is in reality diminished.

(Authors' Abstr.).

Contribution to the Study of Malarial Psychoses. (*Sov. Psychonevr., vol. xii, No.* 5, *p.* 5, 1936.) *Sklar, N. I.*

There is no specific malarial psychosis, but in the course of malaria Bonhoeffer's exogenic forms of reaction may appear as they do in other infectious diseases.

Among 105 cases investigated by the author there were 24 cases of endogenous chronic psychoses and neuroses, also 81 cases of purely infectious acute psychoses (28 of twilight states, 4–7 of delirium acutum, 20 of delirium simplex, 5 manic-depressive delirium, etc.).

In some cases of malaria there exist certain specific traits manifested in general malaise, indisposition, disagreeable bodily sensations (paræsthesiæ, headaches, etc.), followed by a great anxiety with motor excitement.

Among the psychoses appearing after the first paroxysms of malaria acute infectious forms prevail, mostly temporary twilight states.

In the material of the author there was a small percentage of psychopathic disposition (29·4%), that was greater in chronic endogenous psychoses (37·5%) and lesser in cases of acute psychoses (25·4%).

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In acute psychoses occurring during the malarial attacks the provoking agent is the infection itself, the intensity of the process, and also the nature of the reaction, which depends on the form of plasmodium (i.e., if tropical or usual). The intensity and the form of psychoses depend also on the disposition, the constitution of patients and the resistance of the organism. In cases developing after long duration it is the general emaciation that plays a certain role.

In endogenous, chronic psychoses the chief role is played by the disposition, the malaria being only the provocative agent. (Author's Abstr.).

The Curve Pattern of Muscular Contraction and the Tonus Mobility in Psychogenic Pareses. (*Sov. Psychonevr., vol. xii, No. 5, p. 85, 1936.*) *Shirokov, A. A.*

This paper deals with the results of investigation of the functional state of the paretic extremity in psychogenic pareses.

Besides clinical investigation the curves of muscular contraction of the quadriceps muscle were registered, and the muscle tone was determined after the method of V. P. Kusnetzov.

In some patients the palsy was suggested during hypnosis; in other cases there were spontaneous hysterical pareses of the lower extremities. During the paresis the author established a phasic inexcitability in voluntary movements, decrease of excitability (or sometimes complete absence of it) in reflex contractions, and also very low values of the mobility of muscle tonus.

The author concludes that in psychogenic pareses the objective methods of investigation have shown that there is an inhibitory state of the neuro-muscular apparatus of the paretic extremity. (Author's Abstr).

Exophthalmic Goitre and Psychosis. (*Journ. Nerv. and Ment. Dis., vol. lxxxvi, No. 2, p. 152, Aug., 1937.*) *Bram, I.*

Forty-two cases of exophthalmic goitre in association with psychosis are reported.

Sixteen were in men and 26 in women; 12 occurred in surgically untreated patients, the remainder in patients thyroidectomized months or years before coming under observation. Of 30 post-operative cases the psychosis occurred pre-operatively in 10, while in the remaining 20 the mental symptoms appeared for the first time after discharge from hospital.

Though of grave portent, the incidence of mental disorder is not necessarily associated with a high B.M.R.; this is especially true of a psychosis making its appearance after thyroidectomy. The occurrence of a psychosis with Graves's disease makes the prognosis of the prior malady more grave.

The treatment of Graves's disease does not usually cure the psychosis.

T. E. Burrows.

Senile Dementia without Cerebral Atheroma [*La démence sénile sans athéroma cérébral*]. (*Ann. Méd. Psych., vol. xcv* [*i*], *p. 689, May, 1937.*) *Marchand, L.*

A series of six cases of senile dementia without atheroma are investigated. In each case there is a full report upon the clinical findings, the gross morbid anatomy and the histological changes in the brain. Every effort is made only to include cases unobscured by complicating factors, such as syphilis, alcoholism, head injuries, previous mental illness, etc. Dementia senilis without atheroma is excessively rare in the male and consequently all six observations are on females.

Marchand concludes that senile dementia without atheroma is a disease entity and is not merely an exaggeration of normal senile changes. It is due to a primary degenerative process of the parenchymatous nerve-cells, which finally ends in their destruction. There is no evidence of inflammation and there is no circulatory disorder. The neuroglial sclerosis is secondary to the parenchymatous atrophy.

The author believes that in each individual the nerve-cell is endowed with a specific degree of vitality. In the normally developed cell this vitality is sufficient,

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in the absence of noxious exogenous factors, for it to carry on its function to an advanced old age. In senile dementia the nerve-cells fail in their function at the senium. This is due to impoverished vitality of the nerve-cells and is constitutionally determined.

Turning now to the pre-senile dementias Marchand finds no diagnostic sign, either clinical or pathological, which may serve to differentiate Alzheimer's and Pick's disease. At best the dividing line is a quantitative one, the more localized atrophy in Pick's disease. He further points out that all the clinical and pathological changes described in the presenile states are also to be found in senile dementia. Senile plaques, Alzheimer's fibrils and argentophil corpuscles have been found in all three conditions, and therefore cannot be considered pathognomonic of any one of them. He therefore regards the presenile states as exaggerated forms of senile dementia, in which the inherent lack of vitality of the nerve-cells is still more pronounced. He points out that precocious and even juvenile cases of Alzheimer's and Pick's disease have been reported, and also stresses the frequency of a family history of senile dementia in persons suffering from presenile dementia.

STANLEY M. COLEMAN.

Endocrine Disorders in States of Excitation [*Les troubles endocriniens dans les états d'excitation*]. (*Ann. Méd.-Psych., vol. xcv* [*ii*], *p.*15, *June*, 1937.) *Turques, I.*

Reviewing the work done upon the association between endocrine disturbances and maniacal states, it is concluded that this association has been established only in a very small number of cases. Certainly the theory that mania is determined by endocrine changes has not been proved. It is pointed out that the manic-depressive psychosis is actually a heterogeneous group of states with a very diverse ætiology. Consequently it is useless to consider this psychosis as an entity, and research upon the ætiological significance of endocrine unbalance can only be valuable if individual cases of mania are made the subject of study. Again it is necessary to investigate the whole endocrine system, and not the function of an isolated gland.

STANLEY M. COLEMAN.

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4. Neurology.

Encephalography with Anæsthetic Gases. (*Arch. Surgery, vol. xxxiv, pp.* 853–67, 1937.) *Aird, Robert B.*

Nitrous oxide and ethylene are safe agents for encephalography, being non-irritating and leaving no bad after-effects. Ethylene has the advantage of disappearing more slowly and having a slight sedative effect.

JOHN T. MYERS (Chem. Abstr.).

Neurological Symptomatology in Diphtheria. (*Sov. Psychonevr., vol. xii, No.* 5, *p.* 29, 1936.) *Salkan, D. M., and Epstein, P. D.*

As the investigations of the authors have shown, in spite of the existing opinion, the first symptoms of an affection of the nervous system appear quite early (on the third–seventh day) in the course of diphtheria. Only gross symptoms appear late in the course of the disease. The authors admit the elective influence of diphtheria toxin, yet stress the parallelism observed between the intensity of diphtheria and the extensions of lesions within the nervous system. In the development of neurological symptoms it could be noted that the period of deficiency (especially in polyneuritis or multiple neuritis) was often preceded by phenomena of irritation (hyperæsthesiæ), exaggeration of reflexes, general irritability, etc., that appear during the first days of the disease.

As to the symptomatology of diphtheritic lesions, the authors emphasize the pronounced affinity of the diphtheria toxin for the vagus nerve. Paresis of the

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soft palate often does not lead to nasal colouring of language, but it is characterized by simple weakening of active excursions of it.

Among the new symptoms not yet described in the literature, the authors emphasize the frequent occurrence of what they call the foot phenomenon, in which the foot assumes the position as it does in a slight peroneal palsy, with marked hypotonia of muscles. Other symptoms are ptosis of an eyelid, Basedow's and Horner's syndromes. The authors are inclined to consider all those symptoms as belonging to the symptomatology of diphtheria itself and not as complications of it. (Authors' abstr.)

Tumours of the Base of the Brain : Their Relation to Pathological Sleep and Other Changes in the Conscious. (*Med. Journ. Australia, p.* 742, *May* 15*th*, 1937.) *Cox, L. B.*

Six cases of tumour, unassociated with internal hydrocephalus, involving the mid-brain, hypothalamus and thalamus are reported ; each was accompanied by certain changes in the conscious state. These changes included such phenomena as drowsiness, hypersomnia, trance-like conditions, periods of apathy, catatonic states, alterations in disposition, disorientation for time and place, marked loss of memory and mental excitement. In certain cases the removal of the tumour resulted in recovery from the abnormal mental state.

An attempt is made to explain the conditions of lessened consciousness as a disturbance of a sleep mechanism. A working hypothesis is formulated, based on the possible relation of sleep to an inhibitory process.

The relation of conditions of exalted consciousness to the excitation or release from control of mechanisms for the expression of the emotions is discussed.

Attention is drawn to the hypothalamus and basal ganglia in connection with mental illness. T. E. Burrows.

Insulin Response in Acromegaly. (*Bull. Neur. Inst. New York, vol. vi, No.* 2, *p.* 178, *Aug.*, 1937.) *Berg, B. N.*

The author investigated 6 cases of acromegaly. In 3 cases there was a markedly increased resistance to the hypoglycæmic action of insulin. In 2 of these the initial blood-sugar was normal. The third case had a severe diabetes, and the blood-sugar-lowering effect of insulin was markedly diminished and was of the same order as in the other two patients. The " anti-insulin factor " of the gland may or may not be involved in the presence of adenomata affecting the growth factor.
T. E. Burrows.

Manual Reflex. (*Bull. Neur. Inst. New York, vol. vi, No.* 2, *p.* 372.) *Pool, J. L.*

A pathological reflex of the hand is described, consisting of adduction of the thumb upon stimulation of the ulnar portion. It is suggested that the reflex described may be characteristic of disease of the mid-cervical portion of the cord.
T. E. Burrows.

5. Pathology and Biochemistry.

The Influence of the Vegetative Nervous System on Metabolism. (*Vol. jubilaire honneur Parhon* 1934, *pp.* 1–6. *Ber. ges. Physiol. exptl. Pharmakol., vol. lxxxv, pp.* 100–1.) *Abelin, I.*

Three types of substances increase metabolic rate : adrenaline derivatives, the thyroxine group and the recently discovered nitrophenols. 1, 2, 4-dinitrophenol acts by producing either hyperadrenalinemia or hyperthyrosis. Nitrotyrosine and diiododinitrothyronine do not increase metabolic rates. *p*-Nitrophenol resembles, but is weaker in action than, 1, 2, 4-dinitrophenol.
James C. Munch (Chem. Abstr.).

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Phosphorylation and Oxidoreduction of the Glucose Decomposition in the Brain. (*Naturwissenschaften, vol. xxv, pp.* 282–3, 1937.) *Adler, E., Calvet, F., Euler, H. v., and Günther, G.*

Various forms of tissue differ in their ability to split carbohydrates; muscle and its extract decompose glycogen or phosphorylated hexose. Glucose, however, is not decomposed to lactic acid. Brains, embryonic tissue or tumour consume glucose at an even more rapid rate than glycogen. Enzyme preparations from rat brain free of cells and glycolytic in activity were studied. Analogous to the phosphorylase effect of yeast-cells the brain extract transmits labile phosphoric acid groups from adenosine triphosphate to glucose; the triphosphate disappears at equal rate in the presence of co-zymase, pyruvic acid and NaF. Thus phosphorylated hexose is, as usual, the intermediary. For this type of glycolysis the inhibition of lactic acid formation by glyceraldehyde is typical; the inhibition appears in cell-free brain extract, but not in muscle extract. The isolated oxido-reduction hexosediphosphate-pyruvic acid in brain extract is not inhibited by glyceraldehyde.
B. J. C. VAN DER HOEVEN (Chem. Abstr.).

Experiments on the Catarotulin Effect. (*Rec. Trav. Chim., vol. lvi, pp.* 315–29, 1937.) *Westenbrink, H. G. K., and Polak, J. J.*

The O uptake of brain tissue suspended in Ringer-phosphate-pyruvate solution of pigeons in avitaminosis B_1 was measured by the Warburg method, using respiration vessels according to Stern. The addition of vitamin B_1 *in vitro* gave the catarotulin effect which was found without any exception, not only with pigeons showing opistho- or emprosthotonus after curing once or twice by small doses of B_1, but also with pigeons showing these acute symptoms for the first time, or only showing the first disturbances of equilibrium. This increased O uptake was found to be dependent on the time at which B_1 was added. The maximum effect was obtained when B_1 was added before the beginning of the respiration experiment. If B_1 was added to a suspension of the avitaminous brain-tissue in Ringer solution during the estimation of the O uptake the increase is only noted after an induction period of ten minutes. No induction period was observed when pyruvate was added to the tissue suspension which contained B_1. All observations can be explained by the hypothesis that the O uptake of the avitaminous brain in the presence of pyruvic acid is not catalysed by free B_1, but by a compound of B_1 and a still unknown labile substance. C. R. ADDINALL (Chem. Abstr.).

Amount of Phosphorus Compounds in the Brains of Various Animals. (*Biokhimiya, vol. ii, pp.* 216–29, 1937.) *Boldyreva, N. V.*

As a rule the amount of lipoid phosphorus is greater in males than in females. The lipoid phosphorus content of the nerve centres of invertebrates is less than in the brains of vertebrates. Inorganic phosphorus and phosphagen are present in greater amounts in males than in females; the exception is the chicken, which contains more phosphagen than the rooster. The nerve centres of the snail and the brain of the tortoise possess the smallest quantities of phosphagen. This is not typical of invertebrates, as in the cockroach the content of phosphagen is even higher than in several vertebrates. There is no sharp difference in the amount of protein phosphorus found in vertebrates and invertebrates. Birds have comparatively small quantities of protein phosphorus, whereas fish possess considerable quantities of it. H. C. (Chem. Abstr.).

Changes in the Concentration of Potassium and Calcium Ions in Cholinergic and Adrenergic Perfusates Prepared during Excitation of the Vago-sympathetic Nerve-trunks. (*Compt. Rend. Soc. Biol., vol. cxxiv, pp.* 971–2, 1937.) *Gavrilescu, N., and Ionescu, N.*

When frogs are perfused as before described (*C. A.*, vol. xxxi, p. 1444), the potassium content of the perfusing medium increases appreciably when the vagi

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are stimulated. The changes in calcium are too slight and irregular to be of significance. L. E. GILSON (Chem. Abst.).

Free and Combined Acetylcholine of Brain. (*Comp. Rend. Soc. Biol., vol. cxxiv, pp.* 1197–8, 1937.) *Corteggiani, E.*

By simple extraction with cold 0·9% sodium chloride solution (containing eserine) sheep brain yielded about 0·3*v* acetylcholine per grm. If the brain tissue was heated to 70° for three minutes, then cooled and extracted, the yield was about 1 *v*/grm. Extraction of unheated tissue with alcohol or Me_2CO also gave yields of 1*v*/grm. Brain contains acetylcholine in a loosely bound form, which is liberated by heat or by treatment with organic solvents.

L. E. GILSON (Chem. Abstr.).

Effect of Encephalography on Blood-sugar Level of Children. (*Amer. Journ. Med. Sci., vol. cxciii, pp.* 259–64, 1937.) *Bradley, C.*

Following encephalography the blood sugar rises, often to around 200 mgrm.%. It rises rapidly during spinal fluid replacement, reaching the peak within one hour and falls thereafter for several hours. This rise is independent of the anæsthetic used, the clinical condition or age of the child and the amount of fluid withdrawn or air injected. R. C. WILSON (Chem. Abstr.).

The Cholinesterase in Sympathetic Ganglia. (*Journ. Physiol., vol. lxxxix, pp.* 429–37, 1937.) *Brücke, F. Th. v.*

The normal cervical sympathetic ganglion of cats contains a considerable amount of cholinesterase. Its concentration is higher in the sympathetic ganglion than in the normal vagus ganglion or in the cervical portion of the sympathetic nerve. This excess of esterase disappears completely after section of the preganglionic fibres of the sympathetic ganglion. E. D. WALTER (Chem. Abstr.).

The Formation of Acetylcholine by Brain Tissue. (*Journ. Physiol., vol. lxxxix, pp.* 37–8, 1937.) *Stedman, Edgar, and Stedman, Ellen.*

When minced brain tissue is ground with a solution of eserine in chloroform and the mixture then incubated at 37° for 1–4 hours, acetylcholine is formed. Acetylcholine was isolated from such a mixture in the form of its Pt and Au chlorides.

E. D. WALTERS (Chem. Abstr.).

Brain Sphingomyelin in Niemann-Pick's Disease. (*Z. Physiol. Chem., vol. ccxlv, pp.* 163–7, 1937.) *Tropp, C., and Eckardt, B.*

Dry brain powder from a case of Niemann-Pick's disease contained 13·4% sphingomyelin. The fatty acids in this fraction consisted of palmitic, lignoceric and stearic in the ratio 1 : 1·5 : 6. Apart from these facts the analyses agree with those of Klenk. MILTON LEVY (Chem. Abstr.).

Role of Acetylcholine in Transmission of Nervous Stimulation in Striated Muscle. (*Compt. Rend. Soc. Biol., vol. cxxii, pp.* 990–3, 1936.) *Lapicque, L.*

It is suggested that acetylcholine is formed, not between the nerve and the muscle but in the muscle itself, and serves to generalize the stimulations.

B. C. A. (Chem. Abstr.).

Lactacidogen and Lactic Acid in the Surviving Pigeon Brain. (*Ukrain. Biokhem. Zhur., vol. v, pp.* 87–100, 1932.) *Gorodisskaya, H., and Epel'baum, S.*

During two hours in isotonic sodium chloride solution the lactacidogen (I) content of surviving pigeon brain decreases. The addition of starch or glucose is without effect. Lack of oxygen accelerates and electric stimulation retards the disappearance of I. B. C. A. (Chem. Abstr.).

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Influence of Cations and Carbohydrates on the Formation of Inorganic Phosphoric Acid During the Autolysis of Brain Extracts. (*Ukrain. Biokhem. Zhur., vol. v, pp.* 101–12, 1932.) *Gorodisskaya, H., and Epshtein, S.*

In autolyzing extracts of pigeon and cat brains, " summarisches " H_3PO_4 (I) (inorganic H_3PO_4 + creatinephosphoric acid) is formed, but at a constantly diminishing rate. K^+, Na^+, NH_4^+, and Ca^{++} retard, but Mg^{++} accelerates production of I. Addition of glucose, galactose, glycogen, dextrin and starch to the autolysing extract of cat brain retards the reaction, but maltose first retards and then accelerates it. B. C. A. (Chem. Abstr.).

The Glutathione Content of the Blood in Schizophrenia. (*Acta Psychiat. et Neur., vol. xi, p.* 369, 1936.) *Ljungberg, E.*

The writer estimated the glutathione content of the blood by the method of Woodward and Fry. He investigated 41 normals and 93 schizophrenics. Both groups gave the same amount—36·2%. If, however, the schizophrenics were divided into those of less than one year's duration and those of more than one, the results were 40·1% and 32·7% respectively. There was also a significant difference between the older schizophrenias and the normal cases of 3·9%. The investigation showed that in acid filtrates from whole blood there is an oxidizing substance.
G. W. T. H. Fleming.

Observations on Disturbances in the Gastric Secretion in Manic-depressive Psychosis. (*Acta Psychiat. et Neur., vol. xi, p.* 469, 1936.) *Hedström, V.*

The writer investigated 42 cases of manic-depressive psychosis, 34 of which were depressions. He found decreased acidity in many cases amounting to an achlorhydria, increased content of mucus and reduced time of evacuation in the depressions. When the depression clears up normal conditions set in. In a few cases there was noted a high acidity in manic conditions. A relation exists between the acidity values of the gastric juice and the chlorine content in the capillary blood. G. W. T. H. Fleming.

Serum Toxicity in Psychoses. (*Acta Psychiat. et Neur., vol. xi, p.* 665, 1936.) *Wahlström, G.*

The writer tested the effect of serum of psychotics on white mice. He found that serum from schizophrenics and manic-depressives gave fatal results in nearly three times as many instances. Post-mortem examination of the brain revealed increased blood profusion and a more easily crumbling cerebral substance. The ganglion-cells showed vacuolar degeneration. These vacuolar changes also occurred in animals that died after the injection of serum from normal people.
G. W. T. H. Fleming.

Studies in Brain Metabolism. II. Formation of Succinic Acid. (*Biochem. Journ., vol. xxxi, p.* 299, *Feb.*, 1937.) *Weil-Malherbe, H.*

The writer describes a method of succinic acid estimation based on the action of a specific succinic dehydrogenase preparation from ox-heart. This dehydrogenase is inhibited by α-ketoglutaric acid, pyocyanine and phenosafranine. The formation of succinic acid from pyruvic and α-ketoglutaric acids was demonstrated anaërobically with brain slices and with minced brain. Aërobically in experiments with minced brain poisoned with malonic acid, pyruvic, α-ketoglutaric and in some cases acetic acids were found to yield succinic acid. Under the same conditions the respiratory quotient of ketoglutaric oxidation is raised, indicating a specific inhibition of succinic dehydrogenase of roughly 50%. Malonic acid does not lead to succinic acid accumulation with brain slices and with brain mince only if used in certain concentrations. Small amounts of a volatile acid, probably acetic acid, are formed from pyruvic acid by minced brain. Although it is possible

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that α-ketoglutaric acid is an intermediary stage in the series of reactions leading from pyruvic acid to succinic acid, the existence of a second, less important pathway is not excluded, i.e., the formation of succinic acid by the condensation of two molecules of acetic acid as suggested by the original Thunberg-Wieland theory.

G. W. T. H. FLEMING.

Effects of Hydroxymalonate on the Metabolism of Brain. (*Biochem. Journ., vol. xxxi, p. 275, Feb.*, 1937.) *Jowett, M., and Quastel, J. H.*

The writers consider that glucose can be oxidized in brain tissue by a mechanism or mechanisms not involving lactic acid as an intermediary.

Lactic acid is not an intermediary in the oxidation of fructose by brain.

They found that the hydroxymalonate inhibition of glycolysis in guinea-pig brain is very great, but is relatively less in the presence of pyruvate; that with rat brain the inhibition is smaller and little affected by the presence of pyruvate, and that the inhibitions are little affected by a tenfold change in the pyruvate concentration. It is suggested that pyruvic acid is not an intermediary in anaërobic glycolysis in rat brain. Pyruvic acid exerts its accelerating effect on glycolysis in rat brain by acting as a hydrogen acceptor. G. W. T. H. FLEMING.

Oxidation of Aliphatic Amines by Brain and Other Tissues. (*Biochem. Journ., vol. xxxi, p. 286, Feb.*, 1937.) *Pugh, C. E. M., and Quastel, J. H.*

The writers investigated the ability of brain to detoxicate various aliphatic amines. They found that the deamination of butylamine, amylamine, isoamylamine and heptylamine took place in the presence of slices of brain cortex. With the lower amines, propyl, ethyl, methylamine, deamination was less marked or negligible. The respiration of brain cortex (in the presence of glucose) is lowered by the higher amines. The respiration of liver, on the other hand, is increased by the amines which undergo oxidation. Isoamylamine is broken down by brain and liver to yield a substance giving a hydrazone with 2·4-dinitrophenylhydrazine. The possible formation of isoamyl alcohol is indicated by the development of the characteristic odour. The oxidation of the amine appears to occur only under aërobic conditions. An amine-oxidizing system exists in brain and other tissues which is quite distinct from the α-amino-acid-oxidizing system.

G. W. T. H. FLEMING.

The Oxidation of Choline by Rat Liver. (*Biochem. Journ., vol. xxxi, p. 869, June*, 1937.) *Mann, P. J. G., and Quastel, J. H.*

The writers, in view of the use of choline in the treatment of fatty liver and of acetylcholine in the treatment of mental symptoms, investigated the action of rat liver on choline. The increase in oxygen uptake which occurs when choline is added to respiring rat liver slices or extract is due to the oxidation of choline to betaine aldehyde. They also point out the possibility that acetylcholine might arise as a result of a Cannizzaro reaction between betaine aldehyde and acetaldehyde. The betaine aldehyde is still further oxidized, so accounting for the further uptake of oxygen past what would be theoretically expected.

G. W. T. H. FLEMING.

The Mechanism of the Biological Synthesis of Acetylcholine. I. The Isolation of Acetylcholine Produced by Brain Tissue in vitro. (*Biochem. Journ., vol. xxxi, p. 817, May*, 1937.) *Stedman, E. and E.*

The writers used minced brain tissue and not slices. They ground the minced tissue with chloroform before incubation at 37° in the presence of eserine. That acetylcholine is produced under these conditions they showed by its action on eserinized leech muscle, and by the isolation of the double chloroplatinate of choline and acetylcholine, from which they subsequently prepared acetylchloroaurate. The three possible precursors of the acetyl part of the molecule considered were

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acetic, pyruvic and acetoacetic acids. Sodium acetate was found to be without action, and sodium pyruvate had a definite inhibitory action. Sodium acetoacetate, on the other hand, increases the yield of acetylcholine by 50%, and is looked on as the possible precursor. G. W. T. H. FLEMING.

Pyruvic Acid Oxidation in Brain. (*Biochem. Journ., vol. xxx, p.* 2206, *Dec.*, 1936.) *Peters, R. A.*

The writer considers that vitamin B_1 acts specifically on pyruvate oxidase action as follows:

$$\begin{matrix}\text{Lactate} \\ \pm \\ \text{pyrophosphate}\end{matrix} + O \rightarrow \text{pyruvate} + (n)\,O \rightarrow \text{oxidation products.}$$
$$\uparrow$$
$$\text{vit. } B_1$$

The best substrate for this catarotulin effect is pyruvate. He also found that lactate and pyruvate oxidase systems can be separated at acid pH values, and that vitamin B_1 and pyruvate are necessary to ensure stability of pyruvate oxidase at pH 6·6. G. W. T. H. FLEMING.

Arginine Content of Proteins of the Brain in Vertebrates. (*Bull. Biol. Med. Exptl. U.R.S.S., vol. i, pp.* 115–16, 1936.) *Budanova, A. M.*

The arginine (I) content of the proteins varies from 7·4 (hen) to 10·5% (sheep). In the same animal there is no relation between age and I content of brain proteins. B. C. A. (Chem. Abstr.).

Evidence of Selection in the Building-up of Brain Lecithins and Cephalins. (*Journ. Biol. Chem., vol. cxviii, pp.* 131–6, 1937.) *McConnell, K. P., and Sinclair, R. G.*

An adequate diet rich in elaidin was fed to adult female rats during the gestation period and while the young were suckling. The amount of elaidic acid in the lecithins and cephalins of the brain of mature rats so supplied with elaidic acid throughout the entire period of growth from the fertilized egg to maturity is only about a quarter that present in the phospholipides of the liver and muscle of the same animals. Nothing is known as to the factors involved in this selective synthesis of tissue phospholipides. It seems quite evident that the function of most of the brain lecithins and cephalins, whatever it may be, is not that of an intermediary in fatty acid catabolism. A. P. LOTHROP (Chem. Abstr.).

Water Metabolism in Relation to Convulsions. (*Trans. Amer. Neurol. Assoc., vol. lxii, pp.* 64–7, 1936.) *Stone, T. T., and Chor, H.*

The relation between water balance and convulsions claimed by many investigators is not supported. The water metabolism of adult convulsive patients, as revealed by fluid intake and output ratios, blood volume, hematocrit estimations, plasma volume, blood chlorine and body-fluid determinations shows no significant departure from the normal. The incidence of convulsions is not affected by high or low extremes in fluid intake. The convulsions are not attended by significant alterations in the gross amount of body fluid.

MARION HORN (Chem. Abstr.).

A Note on the Identity of the Indophenol-reducing Substances in Brain Tissues. (*Biochem. Journ., vol. xxx, p.* 1883, *Oct.*, 1936.) *Young, F. G.*

The writer points out that the reducing properties of ascorbic acid are greatly modified by the presence of glutathione and other substances present in tissue extracts. There is no reason to believe that the indophenol-reducing substance in crude brain extract is other than ascorbic acid. G. W. T. H. FLEMING.

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Studies on Carbohydrate Metabolism in Nervous and Mental Disorders. III. The Disturbance of the Glucose-tolerance Test Caused by Hypnotics in Clinical Doses. (*Edinburgh Med. Journ., vol. xliv, pp.* 416–20, 1937.) *Tod, H.*

Hypnotics in clinical doses cause a marked effect on the results of the glucose-tolerance test manifested by lowered maximum or shortened hyperglycæmia or both with a markedly decreased hyperglycæmic area. The hyperglycæmic index is generally lowered, but in the case of morphia it tends to be raised.

RACHEL BROWN (Chem. Abstr.).

Acetylcholine Breakdown in the Blood and Cerebro-spinal Fluid. (*Klin. Wochenschr., vol. xvi, pp.* 398–9, 1937.) *Altenburger, H.*

The rectus muscle of the frog sensitized with 0·5 c.c. of 1/10,000 physostigmine was used as the indicator. In blood 0·5 mgrm. of acetylcholine is inactivated after an average of 76 seconds. The spinal fluids from patients with epilepsy and Parkinsonianism showed no characteristic variations from the normal.

WM. MENDELSOHN (Chem. Abstr.).

The Tryptophane Reaction in the Cerebro-spinal Fluid : Its Value in the Diagnosis of Tuberculous Meningitis. (*Lancet,* 1937, *i, pp.* 560–1.) *Spillane, J.*

In 30 out of 32 cases of tuberculous meningitis the spinal fluids gave a positive reaction for tryptophane. The test was negative in 111 fluids from various other diseases. Purulent, hæmorrhagic or xanthochromic fluids gave false positive reactions.

E. R. MAIN (Chem. Abstr.).

A Simple Method for Determining the Quantity of Protein and the Albumin-Globulin Ratio in Cerebro-spinal Fluid. (*Klin. Wochenschr., vol. xv, pp.* 1648–9, 1936.) *Hempel, J., and Giese, L.*

The method depends on the preparation of alcohol albuminate and the evaluation of its turbidity by means of a nephelometer. Five c.c. of 65% alcohol and 0·1 c.c. of cerebro-spinal fluid are placed in the receptacle and the turbidity is read after five minutes. By means of a standard curve, this value is converted into mgrm.% of total protein. This is repeated with 40% alcohol and the value for globulin determined. The difference between these values is that of the albumin fraction. The average protein in normal cerebro-spinal fluid is 23 mgrm.%, and the ratio of globulin to albumin 0·24.

WM. MENDELSOHN (Chem. Abstr.).

The Permeability of the Meninges. (*Bruxelles Med., Dec.* 13, 1936; *Urol. Cut. Rev., vol. xli, p.* 293.) *de Arric, Le Fevre.*

The meninges were impermeable to neoarsphenamine in normal individuals and permeable in twenty-six cases of syphilis, as well as in nine non-syphilitic pathological conditions, including asthenia, dementia præcox, meningeal sarcoma, aphasia, hemiplegia, psychasthenia and polyneuritis from Pb poisoning. The greatest permeability, as measured by determination of As in the cerebro-spinal fluid after intravenous injection of the drug, was found in syphilitic meningo-encephalitis. Malarial crises also increase the permeability. It is suggested that drugs such as hexamethylenetetramine (urotropine) might increase the meningeal permeability and thus add to the effectiveness of arsenical medication. Preliminary testing of this theory permits of no definite conclusions.

MARION HORN (Chem. Abstr.).

Further Observations on the Sulphuric Acid Test in Cerebro-spinal Fluids. (*Trans. Roy. Soc. Trop. Med. Hyg., vol. xxx, pp.* 625–9, 1937.) *Reitler, R., and Pappenheim, E.*

This test was negative for 4 non-syphilitic patients and positive for 13 out of 14 syphilitic patients. The exceptional case may not have been syphilitic. The results of quantitative tests in 27 cases of epidemic meningitis, 4 cases of acute

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poliomyelitis and 3 cases of meningitis of various origin showed a connection between the degree and intensity of the reaction and the lethality of the disease. It seems probable that a positive test is due to the presence of substances derived from nerve tissue, and that the degree of nerve destruction might be indicated by the degree and intensity of the reaction. E. O. WHITTIER (Chem. Abstr.).

Micro-determination of Phosphorus in Cerebro-spinal Fluid. (*Biochem. Zeitschr., vol. ccxc, pp.* 320–6, 1937.) *Tropp, C., Seuberling, O., and Eckardt, B.*

Measure 1·5 c.c. fluid into a 5-c.c. centrifuge tube, add 1·5 c.c. 14% CCl_3CO_2H, keep 3 minutes in a boiling water bath and centrifuge 20 minutes. Transfer 2 c.c. of the supernatant liquid into a 10 c.c. stoppered cylinder and 2 c.c. 7% CCl_3CO_2H into a similar cylinder, and add to each 2 c.c. of reagent (3 parts of a solution of 93 c.c. H_2SO_4 in 1000 c.c. and 1 part of 7·5% sodium molybdate). Mix well, add 1 c.c. of a 1 : 200 freshly diluted 40% $SnCl_2$ in HCl and dilute to volume with *N* H_2SO_4. Examine the colour in the step-photometer using filter S 75. From 0·00125 to 0·01700 mgrm. phosphorus can be determined. For the total phosphorus determination digest 1 c.c. fluid, 2 c.c. water and 0·5 c.c. 14% CCl_3CO_2H with 3 drops H_2SO_4, then proceed as before. For the total acid-soluble phosphorus measure 2 c.c. of the deproteinized filtrate into a combustion flask, and digest with 3 drops H_2SO_4, followed later by 3 drops H_2O_2. With 40% NaOH make alkaline to phenolphthalein (0·1%), and then neutralize with *N* H_2SO_4. Add 1 c.c. of H_2SO_4 solution (93 c.c. in 1 l.), dilute with water to 8 c.c., then add 1 c.c. 7·5% sodium molybdate and 1 c.c. diluted $SnCl_2$ solution. Determine the phosphorus as in the case of the inorganic phosphorus. For the lipoid phosphorus measure 5 c.c. fluid into a 50-c.c. separatory funnel and shake first with 4 c.c. 95% redistilled alcohol, then with 5 c.c. ether, and pour the alcohol-ether through a filter-paper. Repeat the extraction with 5 c.c. ether. Evaporate the lipoid extracts to about 2 c.c., transfer quantitatively to a combustion flask, digest and determine phosphorus as before. S. MORGULIS (Chem. Abstr.).

Proteins of the Spinal Fluid. (*Compt. Rend. Soc. Biol., vol. cxxiv, pp.* 1019–22, 1937.) *Mangelschots, H.*

Most of the material precipitated from human spinal fluid by CCl_3CO_2H is also precipitated by the specific precipitin present in the serum of animals sensitized with human serum-albumen, hence the albumen of spinal fluid is identical with serum-albumen. L. E. GILSON (Chem. Abstr.).

The Pathology of Parkinsonianism. (*Journ. Nerv. and Ment. Dis., vol. lxxxvi, No.* 3, *p.* 265, *Sept.*, 1937.) *Neustaedter, M., and Liber, A. F.*

The authors report 15 cases, 13 of the post-encephalitic and 2 of the idiopathic type.

Cortical alterations, consisting of status cribratus or lacunaris, were found in 4 post-encephalitic and in 1 idiopathic case.

Cytoarchitectonic disturbances were noted in 2 post-encephalitic cases, the Betz cells staining poorly. Neurophagia, cell atrophy and scattered small glia nodules were found in 20 post-encephalitic cases. Punctate areas of demyelinizations in profuse numbers were found in the subcortical white substance in 4 cases of post-encephalitic Parkinsonianism, and in 1 case of the post-encephalitic type with a hemiparesis there was considerable demyelinization in the internal capsule. The large putameno-caudal cells of the striatum were found in various degrees of destruction without exception in all cases. The small cells were slightly affected in 1 idiopathic case and in 3 post-encephalitics considerably, and markedly reduced in number and exhibiting considerable neurophagia. In still another post-encephalitic case they stained rather poorly.

The globus pallidus exhibited marked damage in all cases. There was a paucity of cells and the few remaining were shrunken, some filled with lipoid, others swollen

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with eccentric nuclei and containing a darkly granular substance or appearing in narrow band forms mostly as cell shadows.

In addition there appeared darkly basophilic staining concretions loosely scattered throughout the parenchyma and also infiltrating the media of the blood-vessels, and scattered about in droplets along capillaries in all but one post-encephalitic case. These concretions seemed to be fairly limited to this ganglion. They proved to consist mostly of colloid iron.

The red nucleus was affected in all cases except one of post-encephalitic type.

The substantia nigra was severely affected throughout in 13 post-encephalitic cases. The cells of the dentate nucleus were in various stages of degeneration in 12 post-encephalitic and 1 idiopathic case.

In the medulla the olives were markedly affected in 13 cases and were fairly preserved in 2 post-encephalitic cases.

Anterior horn-cell destruction in the cervical region was found in 3 post-encephalitic cases. The cerebellum, thalamus, hypothalamus, locus cœruleus, substantia innominata of Reichert and the mammillary bodies were fairly intact in all cases.

T. E. BURROWS.

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6. Pharmacology and Treatment.

Pharmacology of Phenylisopropylamine (Benzedrine). (*Journ. Pharmacol., vol. lx, pp.* 56–68, 1937.) *Detrick, L. E., Millikan, R., Modern, F. S., and Thienes, C. H.*

dl-phenylisopropylamine stimulated the central nervous system of anæsthetized animals, causing struggling and increase in rate and depth of respiration. It had a pressor action in dogs, cats and rabbits. The potency was 1/100–1/500 that of adrenaline. Tachyphylaxis was marked. The pressor effect was moderately decreased by cocaine or ergotamine. In concentrations of 10^{-4} or higher, dl-phenylisopropylamine caused contraction of all smooth muscles. In the cat's ileum and duodenum a temporary inhibition preceded the contraction, which was decreased by nicotine and prevented by atropine. Atropine did not alter the contraction of cat or rabbit intestine produced by dl-phenylisopropylamine, but abolished that of guinea-pig intestine. Ergotamine abolished the action of adrenaline on cat duodenum, but not the action of dl-phenylisopropylamine, while hydrastinine inhibited the action of both dl-phenylisopropylamine and adrenaline and yohimbine had a negligible effect in either case.

L. E. GILSON (Chem. Abstr.).

Treatment of Narcolepsy with Benzedrine Sulphate. (*Journ. Kansas Med. Soc., vol. xxxviii, pp.* 208–9, 1937.) *Tihen, Henry N.*

Oral administration of benzedrine sulphate, in 10-mgrm. doses twice daily, maintained two cases of narcolepsy practically symptom-free without any untoward effects. Both cases were refractory to other forms of treatment, i.e., ephedrine, pituitary extract, thyroid extract, ketogenic diet and potassium.

MARION HORN (Chem. Abstr.).

The Determination of Barbiturates in Blood and Urine by a New Method. (*Journ. Pharmacol., vol. lix, pp.* 379–92, 1937.) *Brundage, J. T., and Gruber, C. M.*

Haden blood-filtrate (cf. *C.A.*, 17, 2895) or acidified diluted urine is shaken with acid-washed activated charcoal to adsorb the barbiturate. From the charcoal the barbiturate is extracted with a mixture of equal parts of petrified ether and Et_2O, the solvent evaporated, the residue dissolved in $CHCl_3$ and the barbiturate determined colorimetrically by the method of Koppanyi, *el al.* (*C.A.*, 28, 826^6). The depth of colour produced by equimolar solutions of different barbituric acids dissolved in $CHCl_3$ with the above $Co(OAc)_2$-isopropylamine test was not found to

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be related to the molecular weight of the barbiturate. Ortal Na, Na pentobarbital and Na amytal, when injected intravenously, leave the blood-stream rapidly. Only a very small fraction of the amount can be found in the blood one minute later. The substance eliminated in the urine seems to be a degradation product and not the barbiturate itself. Anæsthesia never resulted in mice when the extracted material was injected intraperitoneally.

L. E. GILSON (Chem. Abstr.).

A New Method for the Determination of Barbital. (*Arch. Exper. Path. Pharmakol., vol. clxxxiii, pp.* 70–6, 1936.) *Sack, G.*

Five parts of urine (10–20 c.c.) plus 2 parts 30% Pb acetate plus 1 part 1% AcOH are filtered and washed three times with 1% AcOH. The yellow filtrate is shaken four times with 10–20 c.c. ether. The small amount of aqueous solution is added to 5 c.c. distilled water and 10 c.c. of K dichromate-sulphuric acid (2 grm. K_2CrO_4 plus 20 c.c. concentrated H_2SO_4 diluted to 100 c.c.) and heated for half an hour on a water-bath. After cooling, the dichromate is reduced by addition of 0·3–0·5 c.c. perhydrol. The resulting green complex chromesulphate and the K_2SO_4, which are insoluble in water, are shaken four times with 10–20 c.c. ether. The ether extract is again evaporated and the remaining aqueous solution is evaporated to dryness. The residue is dissolved in 5–8 c.c. absolute alcohol, filtered, washed with alcohol and evaporated to dryness. The residue is taken up with 10 c.c. water, filtered, washed with water and finally shaken four times with 10–20 c.c. ether. The dried residue from the ether extract contains the pure barbital. The limit of error of the method is 5% if the amount present is not less than 2 mgrm.

EDWARD EAGLE (Chem. Abstr.).

The Relation of Barbital and Phenobarbital to Granulocytopenia. (*Journ. Amer. Pharm. Assoc., vol. xxvi, pp.* 483–5, 1937.) *Kopet, J. C., and Goodrich, F. J.*

Drugs similar to amidopyrine, having an amine group attached to a C_6H_6 nucleus in their structural formula, are apparently the cause of this condition. Barbital and phenobarbital do not cause a permanent decrease in the number of circulating granulocytes in the peripheral blood of normal rabbits.

A. POPINEAU-COUTURE (Chem. Abstr.).

Ephedrine as an Antidote for Acute Barbituric Acid Poisoning. (*Riforma Med., pp,* 1643–50, 1934.) *Abbatecola, B.*

The lethal dose of sodium barbital injected intravenously into rabbits was 500 mgrm./kgrm. Injection of 300 mgrm./kgrm. produced narcosis for some hours. Intravenous injection of less than 1 mgrm. of ephedrine per kgrm. had no effect; 2–10 mgrm./kgrm. lengthened the barbital narcosis to 10–14 hours; 30–60 mgrm./kgrm. showed no greater effects; doses larger than 60 mgrm./kgrm. shortened the period of narcosis. Ephetonine behaved like ephedrine. The respiratory centre was stimulated by ephedrine. After a fatal dose of barbital, ephedrine did not show significant antagonism.

JAMES C. MUNCH (Chem. Abstr.).

The Pharmacology of the Vegetative Nervous System. I. Mechanism of Action of Barbital and Phenobarbital. (*Rass. ter. pat. clin., vol. vi, pp.* 526–49, 1934.) *De Biasio, B.*

Circular and pendulum movements of the isolated rabbit and rat intestines were recorded. In the concentrations of 500–1000 parts per million phenobarbital was a stronger depressant than barbital. After $BaCl_2$ the rhythmic contractions were more strongly depressed. Circular movements were depressed before longitudinal. Reversibility of phenobarbital action is associated with disturbances in the colloidal composition of the intestinal muscles.

JAMES C. MUNCH (Chem. Abstr.).

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Certain Pharmacological Actions of the Newer Barbituric Acid Compounds. (*Amer. Journ. Obstet. Gynecol., vol. xxxiii, pp.* 729–44, 1937.) *Gruber, C. M.*

The substitution of S for the O in the malonylurea ring of sodium pentobarbital has changed the actions of the drug enormously. An objectionable feature of all barbiturates in obstetrics is the high percentage of cases showing extreme restlessness and maniacal symptoms. They depress the respiratory centre and death may result. The heart-muscle is depressed. The rapid injection of any of the barbiturates causes a sudden fall in the blood-pressure. A depressant action is exerted on all smooth muscles, especially when larger doses are given.

R. C. WILLSON (Chem. Abstr.).

Acute Barbiturism of the Hallucinatory and Pseudo-cerebellar Type. (*Arch. Soc. Sci. Med. Biol. Montpellier et Languedoc, vol. xvii, pp.* 54–7, 1937.) *Euzière, Vidal, Lafon and Quet.*

Hallucinations, confusion and cerebellar involvement, persisting four days, were observed in a man 38 years old three days after he had swallowed thirty-seven tablets of 5-ethyl-5-phenylbarbituric acid (gardenal), each containing 0·10 (grammes ?). Therapy was by injections of camphorated oil.

MARION HORN (Chem Abstr.).

Action of Phenobarbital on the Adrenaline-sensitive Vasodilators. (*Compt. Rend. Soc. Biol., vol. cxxiv, pp.* 1293–6, 1937.) *Raymond-Hamet.*

Phenobarbital, sparteine and cocaine increase the hypertensive action of adrenaline. Cocaine increases, while phenobarbital and sparteine invert, the hypotensive action of dihydroxyphenylethanolethylamine; hence phenobarbital resembles sparteine in its action. L. E. GILSON (Chem. Abstr.).

Physiology and Pharmacology of the Autonomic Nervous System. XXIV: The Action of Amines on the Nictitating Membrane and the Effect of Cocaine and Denervation on their Action. (*Arch. Intern. Pharmacodynamie, vol. lv, pp.* 190–222, 1937.) *Bacq, Z. M.*

The contraction of the nictitating membrane of the dog and cat was used to test the relative activities of various adrenaline-like compounds. *d*- and *l*-adrenaline, arterenol, epinine and adrenalone have a very similar action in contracting the membrane. Dihydroxyephedrine and ethylnorsuprarenine inhibit the membrane. The degree of sensitization by cocaine varies from × 5 for adrenaline to × 50 for epinine. The monophenolic amines, such as tyramine, icoral, *p*-hydrozy-*m*-methylphenylaminoproponol and hordenine have less rapid but more prolonged action. Cocaine does not sensitize for the action of tyramine and *p*-hydrozy-*m*-methylphenylaminopropanol, but does for the others. Denervation sensitizes generally for the group. The non-phenolic amines produce less prolonged contractions, and with the exception of amino-l-phenylacetone they are diminished by cocaine. Substitution in the ring of NH_2-, CH_3- OCH_3- or Cl- or the addition of another pH group diminishes the action, and cocaine and denervation have variable effects. Me-, Et- and isoamylamine cause contractions which are sensitized by cocaine and by denervation. The action of β-tetrahydronaphthylamine is greater than that of α- or β-naphthylaminopropanol. Their actions are diminished by cocaine and denervation. M. L. C. BERNHEIM (Chem. Abstr.).

Pharmacology of the Sympathetic Inhibitory System. I. Action of Methoxy-2-iodo-5-phenoxyethyldiethylamine (1081 *F*). (*Arch. Intern. Pharmacodynamie, vol. lv, pp.* 223–32, 1937.) *Bovet, D., and Simon, A.*

It paralyses the sympathetic inhibitory fibres in the cat, it prevents the relaxation of the intestine, stomach and the non-gravid uterus which is usually caused by splanchnic stimulation and by adrenaline.

M. L. C. BERNHEIM (Chem. Abstr.).

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Action of Nicotine on Nerve Excitability and a New Interpretation of Langley's Phenomenon. (*Compt. Rend. Soc. Biol., vol. cxxiv, pp.* 1290–3, 1937.) *Chauchard, P.*

By the application of a 0·03% solution of nicotine (instead of the 1% solution used by Langley and many others) to a sympathetic nerve it was found that nicotine has an elective action on the pre-ganglion fibres. Their chronaxie first decreases, then increases, then the fibres become inexcitable. Post-ganglionic fibres are not affected by the diluted nicotine solution, but are made inexcitable by a 0·7% or stronger solution. Langley's phenomenon is thus explained without the necessity of assuming a special sensitivity of the ganglia.

L. E. Gilson (Chem. Abstr.).

Action of Mescaline on the α-waves (Berger Rhythm) in Man. (*Compt. Rend. Soc. Biol., vol. cxxiv, pp.* 1296–9, 1937.) *Chweitzer, A., Geblewicz, E., and Liberson, W.*

The injection of 0·165 grm. mescaline sulphate caused a decrease in the amplitude of the α-waves and an increase in the periods of quiet of the electric encephalogram. The effects persisted several days. During the first few hours the absence of α-waves seemed to occur at the time the patient was having visions.

L. E. Gilson (Chem. Abstr.).

Experimental Determination of the Anticonvulsant Properties of Some Phenyl Derivatives. (*Science, vol. lxxxv, pp.* 525–6, 1937.) *Putnam, T. J., and Merritt, H. Houston.*

Phenyl, cresyl and tolyl sulphonates, benzoates, ketones and esters of carbamic, malic and barbituric acids and hydantoins were tested. Of these diphenylhydantoin, acetophenone and benzophenone were found to be the most effective anti-convulsants with the least relative hypnotic effect. An electric set-up for determining the threshold of convulsions in an experimental animal is described.

T. Laanes (Chem. Abstr.).

Pathological and Histological Study of the Nerve-centre System in Morphinism. IV. Experiments on Guinea-pigs. (*Journ. Orient. Med., vol. xxi, pp.* 79–80, 1934.) *Hayashi, A.*

Like dogs, cats and rabbits, guinea-pigs acutely poisoned with morphine showed no detectable changes in the pia mater, but showed an increase in lipoids during chronic poisoning. Hyperæmia and multiple hæmorrhagic areas followed both types of poisoning. Acute poisoning produced slight pathological changes in the brain-stem and corpus striatum, and possibly in the cerebellum, which were not observed after chronic poisoning.

V. Experiments on White Rats. (*Ibid., pp.* 81–2.)

The pathological changes in rats corresponded with those observed in guinea-pigs.

VI. Experiments on Mice. (*Ibid., pp.* 83–4.)

The pathological changes in mice corresponded with those observed in guinea-pigs.

VIII. Experiments on Domestic Fowls and Frogs; Conclusions. (*Ibid., pp.* 85–7.)

Fowls showed brain changes similar to those observed in guinea-pigs. The brains of frogs, exposed to chronic or to acute morphine poisoning, showed very little pathological change. The lipoid content of the brains of frogs was not increased by chronic or acute morphinism. James C. Munch (Chem. Abstr.).

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Nystagmic Head Deviation : A Selective Action of an Aliphatic Bromine Compound. (*Journ. Pharmacol., vol. lix, pp.* 253–9, 1937.) *Loewe, S.*

In the albino mouse, but not in other animals, monobromobutylurea produces head and body nystagmus, a phenomenon consisting of a periodic tonic lateral deviation and intercurrent rapid retrogradation. The locus of attack of the drug is a highly central apparatus for postural tonus. None of a series of closely related compounds produced the effect. The antagonistic or synergistic influence of various other drugs was studied. L. E. Gilson (Chem. Abstr.).

Physiologic Effects of Acetyl-β-methylcholine (Mecholyl) and Its Relationship to Other Drugs Affecting the Autonomic Nervous System. (*Amer. Journ. Med. Sci., vol. cxciii, pp.* 198–214, 1937.) *Myerson, A., Loman, J., and Dameshek, Wm.*

Mecholyl (I) was given subcutaneously alone and in combination with atropine, sodium amytal (II), benzedrine (III) and adrenaline (IV) to two paretics and 18 cases of various types of dementia præcox. In 30-mgrm. doses I produced its typical effects. The combined effect of II and I on the blood-pressure is additive, the resultant decrease being very great. II decreased the effect of I on perspiration. When IV and I are both given subcutaneously, the effect of I on the blood-pressure overshadows that of IV. When IV is given intravenously, its action on the blood-pressure overshadows that of I. IV is unable to check or modify the other visible effects of I. The same statement holds true for III, its prolonged pressor action being temporarily nullified by I.

R. C. Willson (Chem. Abstr.).

Brilliant Vital Red as an Anticonvulsant. (*Trans. Amer. Neurol. Assoc., vol. lxii, pp.* 139–42, 1936.) *Cobb, S., Cohen, M. E., and Ney, J.*

Neutral red (I) and brilliant vital red (II) have been found to act as anti-convulsants in children and in experimental animals. In mice injected intraperitoneally with I convulsions induced by triphenyl phosphite were delayed and less severe, those due to camphorated oil (III) were delayed, and those due to picrotoxin were not delayed and were more severe. Rabbits injected intravenously with II until they became conspicuously red in the scleræ, skin and mucosa proved especially resistant to the convulsive action of III and of strychnine sulphate. Convulsions were diminished in number and severity in five out of six children suffering from frequent convulsions, and treated with intravenous injections of 1% II until the skin became pink. In the sixth case the number of convulsions was increased after administration of II. Marion Horn (Chem. Abstr.).

Administration of Small Doses of Bromine to Neurotic Patients. (*Byull. Vsesoyuz. Inst. Eksptl. Med., Nos.* 9–10, 8–12, 1935.) *Vainberg, I. S., and Birman, B. N.*

Bromine therapy has been used by Pavlov's laboratories on animals having neurotic disturbances. Large doses of bromine were effective in cases of the "strong" types of nervous system (predominance of excitatory processes), while small doses gave good results in the "weak" types (predominance of inhibitory process). This method was applied by the authors to forty-six neurasthenic patients who were given bromine for a period of three to six weeks. Small doses were most effective (two tablespoonfuls of a 0·1–0·25% solution daily), especially in eliminating insomnia. Bromine was ineffective in cases exhibiting no insomnia. In two-fifths of the cases the effects of bromine persisted for a few months after its administration was discontinued, while in the others the beneficial effects lasted only as long as bromine was administered. In most cases the beneficial clinical symptoms were accompanied by corresponding increases in the blood bromine.

S. A. Corson (Chem. Abstr.).

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Action of Different Doses of Bromine on Inhibition in Monkeys of the Excitable Type. (*Byull. Vsesoyuz. Inst. Eksptl. Med., Nos.* 9–10, 14–15, 1935.) *Kaminskii, S. D., and Matorov, F. D.*

Bromine was administered to two excitable monkeys 45 minutes before the experiment (attempts to establish conditioned differentiation to a metronome and a red light). After a few days small doses (0·5–1·0 grm.) produced an increase in the inhibitory process, thus strengthening differentiation and calming the animals. Differentiation disappeared when the bromine adminstration was discontinued, or when larger doses of bromine (2·5 grm.) were used.

S. A. Corson (Chem. Abstr.).

Toxicology and Pharmacology of Mescaline. (*Rass. ter. pat. clin., vol. vi, pp.* 577–94, 1934.) *De Nito, G.*

Administration of 20–30 mgrm. of mescaline sulphate weakened the reflexes of frogs. After guinea-pigs were given 100–200 mgrm. per kgrm., sensitivity to pain was increased and restlessness developed. Dogs showed no change following faradic stimulation after mescaline; 40 mgrm. per kgrm. slowed the heart and caused a fall in blood-pressure, which was not effected by vagotomy or atropinization. The tonus of smooth muscle was decreased by concentrations of 20 parts per million. The respiratory centre was depressed in warm-blooded animals, leading to death. James C. Munch (Chem. Abstr.).

The Action of Eserine and Related Compounds and of Acetylcholine on the Central Nervous System. (*Journ. Physiol., vol. lxxxix, pp.* 165–97, 1937.) *Schweitzer, A., and Wright, Samson.*

Cats were used (usually under chloralose anæsthesia) as experimental animals; the knee-jerk is used as the type somatic reflex. Eserine (0·03–0·2 mgrm. per kgrm. body-weight) markedly increases the knee-jerk and general reflex excitability. Larger doses (up to 0·75 mgrm. per kgrm.) produce an increase in tone and convulsive movements. Atropine (0·5–2 mgrm. per kgrm.) depresses the knee-jerk. Prostigmine and Stedman's "meta" compound (the methyl carbamic ester of *m*-hydroxyphenyltrimethyl ammonium iodide) depress the knee-jerk owing to a direct inhibitory action on the spinal cord. The inhibition is abolished by eserine. Prostigmine, acetylcholine and the "meta" compound abolish convulsions induced by strychnine. Pilocarpine and arecoline are purely convulsant drugs and have little heightening action on the knee-jerk. Acetylcholine (0·03 mgrm. per kgrm. or over) usually depresses the knee-jerk in unatropinized animals. Eserine increases the inhibitory effects of acetylcholine on the knee-jerk. Acetylcholine inhibits the knee-jerk in the spinal animal, and when the hind limbs are separately perfused or have their circulation occluded. E. D. Walter (Chem. Abstr.).

Technique of Prolonged Narcosis with Cloetta's Mixture. (*Sov. Psychonevr., vol. xii, No.* 6, *p.* 65, 1936.) *Richter, G. E., and Stepanov, P. N.*

The authors consider as contra-indications for the treatment: (1) Active forms of pulmonary tuberculosis; (2) tuberculosis of other organs, including osteal tuberculosis; (3) chronic and acute bronchitis; (4) all pulmonary affections with destruction of lung tissue (abscess, gangrene); (5) acute inflammatory diseases of pleura and lungs; (6) emphysema of the lungs; (7) decompensated and slightly compensated cases of valvular disease of the heart; (8) myodegeneration of the heart and cardiocirrhoses; (9) nephritis, nephrosis, nephrosclerosis; (10) diseases of the liver and bile-passages; (11) acute and sub-acute diseases of the digestive tract; (12) all acute and subacute conditions of the urinary and sexual paths.

The intensity of narcosis ought to correspond with the severity of the disease The body temperature must be taken every two hours and the values obtained used for the regulation of further doses. A slight fever up to 37·5° C. is quite normal

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for prolonged sleep, but fever above 38° C. may be a sign of a commencing complication and make the physician careful. The complications that are observed depend partly on the phenotypic properties of the patient and partly on the narcosis itself. Among them the most menacing are the pulmonary complications, which are liable to cause sudden death. In one case there was a depression of the vagus, with small and frequent pulse. In some cases irritation of the kidneys and certain disorders of metabolism were observed.

A deficiency of the original methods of Cloetta-Maier is, in the opinion of the authors, feeding by glucose alone, which is liable to cause mineral hunger and thus lead to complications. To prevent this the author recommends the additional administration of physiological solution and of Tirode's fluid. Feeding *per os* ought to be avoided.

The average duration of sleep should be ten or twelve days.

(Author's Abstr.)

Delayed Death and Mortality following Cardiazole, Ephedrine and Cardiazole-ephedrine Combinations after Lethal Doses of Sodium Barbital. (*Arch. Exper. Path. Pharmakol., vol. clxxxiii, pp.* 138–45, 1936.) *Gros, O., and Hofmann, H.*

Cardiazole injections (40 mgrm./100 grm.) can prevent the onset of respiratory failure in the rat following lethal doses of Na barbital (40 mgrm./100 grm.). Delay in cardiazole administration renders the antidote action ineffective. Combination of cardiazole (16·5 mgrm./100 grm.) and ephedrine (1·5 mgrm./100 grm.) will prevent death from 50% above the lethal dose of Na barbital; cardiazole alone will not do so. EDWARD EAGLE (Chem. Abstr.).

The Effect of Cardiazole and Ephedrine on the Blood Pressure of Animals Previously Treated with Narcotics. (*Arch. Exper. Path. Pharmakol., vol. clxxxiii, pp.* 146–58, 1936.) *Hofmann, H.*

Cardiazole (0·5 mgrm./100 grm.) increases the blood-pressure of rats. Animals previously treated with paraldehyde or chloral hydrate required 1 mgrm./100 grm. of cardiazole; following Na barbital, the minimal dose of cardiazole is 2·0 mgrm./100 grm. Ephedrine (0·05–0·1 mgrm./100 grm.) increases the blood-pressure of rats, but after Na barbital administration, the smallest effective dose of ephedrine is 0·2 mgrm./100 grm. Following Na barbital, marked increase in blood-pressure is obtained by combined injection of cardiazole (5 mgrm./100 grm.) and ephedrine (1 mgrm./100 grm.). EDWARD EAGLE (Chem. Abstr.).

The Effect of Antipyretics on the Action of Soporifics. (*Arch. Exper. Path. Pharmakol., vol. clxxxiii, pp.* 236–55, 1936.) *Girndt, O., and Huhn, O.*

Antipyretics exert an antagonistic action, arresting the onset of narcosis and decreasing the magnitude of respiratory fall and depth of narcosis. It was also found that bromural arrests the stimulating action of pyramidone.

EDWARD EAGLE (Chem. Abstr.).

The Stimulating and Restorative Properties of Methylimidazole, Including Comparison with the Action of Cardiazole and Coramine. (*Arch. Exper. Path. Pharmakol., vol. clxxxiii, pp.* 478–99, 1936.) *Axmacher, Fr.*

The average convulsive dose of methylimidazole in rabbits is 45 mgrm./kgrm.; the average lethal dose is 75 mgrm./kgrm. Anæsthesia produced by 1 grm. urethan/kgrm. is diminished in depth, but prolonged by methylimidazole and is followed by a prolonged increase in respiratory rate. The anæsthesia produced by 0·18–0·24 grm. barbital/kgrm. is hardly affected by methylimidazole, although a few reflexes are improved. There was no prolongation of anæsthesia nor a marked increase in respiratory rate. The convulsive and lethal doses of methylimidazole are raised by urethan or barbital anæsthesia.

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The Circulation Under the Action of Methylimidazole. (*Ibid., pp.* 500–8.)

The blood-pressure of cats and rabbits under urethan anæsthesia is raised by methylimidazole; larger doses, however, lower the blood-pressure. Methylimidazole has no direct action on the cardiac activity of rabbits; the changes in minimum volume are indirect. EDWARD EAGLE (Chem. Abstr.).

Some Effects of Bulbocapnine on the Peripheral Vascular System. (*Journ. Pharmacol., vol. lix, pp.* 458–67, 1937.) *Leese, E. Chester, and Fogelberg, Alma.*

In etherized dogs the intraperitoneal injection of 3–60 mgrm./kgrm. of bulbocapnine produced an average decrease of 37% in arterial pressure and 50% in venous pressure within 30 minutes. It is believed that bulbocapnine interferes with the peripheral vasomotor mechanism because adrenaline and acetyl-methylcholine chloride are less effective in altering arterial pressure after bulbocapnine than before, while the effectiveness of pitressin and histamine is increased or unchanged. Cardiac decompensation is excluded as a factor. Possibly the static pulse of catatonia from bulbocapnine is due to a decreased irritability of the vasomotor mechanism. L. E. GILSON (Chem. Abstr.).

The Action of Histamine on the Cerebral Blood-vessels. (*Klin. Wochenschr., vol. xvi, pp.* 236–7, 1937.) *Bruch, H.*

The subject used was a man with a traumatic opening in the temporal bone through which the cerebral pulse could be observed. One c.c. of a 1 : 200,000 solution of histamine was given intravenously. The pulse excursions, which were recorded on a kymograph, immediately became larger and the rate increased from 60 to 80 per minute. A simultaneous tracing from the upper arm showed a peripheral vascular dilatation at a much later time. These differences may be due to either the greater sensitivity of the cerebral blood-vessels or the more rapid blood circulation in the brain. W. MENDELSOHN (Chem Abstr.).

The Action of Mid-Brain Narcosis on Blood Iodine in Basedow's Disease. (*Wien. Arch. Inn. Med., vol. xxx, pp.* 135–46, 1937.) *Fenz, E., and Uiberrak, K.*

Narcosis of the mid-brain brought about by the administration of prominal or barbital produces in individuals with Basedow's disease and hyperthyroidism a more or less marked lowering of the iodine content of the blood. In individuals with normal metabolism, on the contrary, there is no lowering or a very moderate one of the iodine content of the blood, and in a case of myxœdema no lowering at all. The lowering of the iodine content of the blood after mid-brain narcosis is the greater the higher the iodine content of the blood before the narcosis. A parallelism exists between the height of basal metabolism and the iodine content of the blood. The lowering of the iodine content of the blood in individuals with Basedow's disease after mid-brain narcosis indicates that mid-brain narcosis causes a decrease in production of secretion by the thyroid gland.

HARRIET F. HOLMES (Chem. Abst.).

Art and Therapy in the Mental Disturbances of Children. (*Journ. Nerv. and Ment. Dis., vol. lxxxvi, No.* 3, *p.* 249, *Sept.*, 1937.) *Bender, L.*

Graphic art is a means of establishing rapport with children who are not spontaneously expressive; who have speech or language difficulties, who are reluctant to discuss their intimate problems or who are taciturn or withdrawn.

It is a means of obtaining insight into the child's unconscious life. Often, however, it seems that the mere expression of unconscious fantasies is of psychotherapeutic value.

There appears to be evidence that the graphic expression of the aggressive impulses in the young child and often also of sexual drives in young adolescents has definite psycho-therapeutic value. Art classes are a socializing force in many

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ways. There is also a socializing value in the fact that the child has produced something which he can give to the group or to his admired psychotherapist or teacher in order to help win their admiration and love. There is undoubtedly also therapeutic value in the opportunity to express the impulses for motor activity, and in the creation and experimentation with forms regardless of their ideational content. For the very young child, the mentally defective, the severe neurotic or schizophrenic child, who is struggling to understand the world of reality, a pencil paper play probably gives considerable satisfaction to impulses for patterned activity.

Art productions have given a particularly valuable clinical record of certain types of behaviour difficulties, of their progress and often of their improvement under psycho-therapy. T. E. BURROWS.

Fever Treatment of Dementia Præcox with Sulphur in Oil. (*Journ. Nerv. and Ment. Dis., vol. lxxxvi, No. 2, p. 162, Aug., 1937.*) *Shapiro, L. B., and Read, C. F.*

The authors report on the results of treatment of 103 recent cases: Remission 11, marked improvements 19, amelioration of symptoms 35, no change 38.

Their experience shows that sulphur in oil therapy is a useful adjunct in the treatment of both recent and chronic cases.

The value of its use in the management of acutely excited, perplexed and bewildered cases has been strikingly demonstrated.

Cases of this type that respond usually begin to show improvement during the course of treatment. T. E. BURROWS.

Syphilis and Neurosyphilis Treated by Electro-pyrexia. (*Med. Journ. Australia, p. 895, June 12, 1937.*) *Prior, G. P. V.*

The author reviews the literature and reports on the treatment by means of electric heat of 67 patients suffering from various forms of syphilis; 38 were treated with diathermy and the others with inductothermy.

The patients were not selected, and the results on the whole were favourable. Electrical pyrexia seems to bring about changes in the serological reactions in a much shorter time than does malaria. A seemingly greater influence on the serological reactions was obtained with diathermy as compared with inductothermy.

The total number of hours of treatment above a given temperature, whether produced by malaria or other means, does not seem to have any direct bearing upon the clinical and serological results. T. E. BURROWS.

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CONTENTS FOR SEPTEMBER, 1937

Original Articles— PAGE

Some Observations on the Care of the Insane: Presidential Address; by *Douglas McRae, M.D., F.R.C.P.E.* 489

The Circulatory System and Mental Disorder: A Symposium—

Introduction: Some Remarks on the Physiology of the Cerebral Circulation; by *F. L. Golla, F.R.C.P.* 505

The Histopathological Criteria of Vascular Disturbances in the Brain; by *Alfred Meyer, M.D.* [Abstract] 509

The Relative Vascularity of Different Areas of the Mammalian Brain; by *A. Colin P. Campbell, M.B., Ch.B., M.R.C.P.Ed.* [Abstract] . 510

Cerebral Ischæmia and Mental Disorder; by *F. A. Pickworth, M.B., B.S., B.Sc., A.I.C.(Exam.)* 512

The Circulatory Factor in the Pathogenesis of Mental Disorder; by *E. Krapf, M.D., F.R.F.P.S.G.* 534

Psychological Implications of Func ional Circulatory Disorder; by *E. Guttmann, M.D.* 542

Psychoses Associated with Hypertension, Arteriosclerosis and Heart-failure; by *W. Mayer-Gross, M.D.* 551

Clinical Note—

A Fatal Case of *Status epilepticus*, with Post-mortem Findings; by *A. M. Wyllie, M.B., Ch.B., B.Sc.* 568

Reviews 571

Administrative Psychiatry; by *W. A. Bryan, M.D.*—Treatment in Psychiatry; by *Oscar Diethelm, M.D.*—Research in Dementia Præcox; by *Nolan D. C. Lewis, M.D.*—Principles and Practice of Recreational Therapy for the Mentally Ill; by *J. H. Davis, M.A.*—The Intellectual Functions of the Frontal Lobes; by *R. M. Brickner, M.D.*—The Self in Psychology; by *A. H. B. Allen.*—The Logical Structure of Science; by *A. Cornelius Benjamin.*—Towards a Law of Creative Thought; by *Rosamond E. M. Harding.*—Mitogenetic Analysis of the Excitation of the Nervous System; by *Prof. A. G. Gurwitsch*; translated by *G. and F. Engel.*—L'Hystérie et l'Évolution Humaine; by *M. Dide, M.D.*—The Psychology of Belief; by *H. Lundholm, Ph.D.*—Freud and Marx; by *R. Osborn.*—The Background of Spiritual Healing; by *A. Graham Ikin, M.A., M.Sc.*—The Unknown Murderer; by *Theodor Reik.*

Epitome of Current Literature 582

1. Anatomy and Physiology.—2. Psychology and Psychopathology.—3. Psychiatry.—4. Neurology.—5. Pathology and Biochemistry.—6. Pharmacology and Treatment.

CONTENTS OF SUPPLEMENT

The Royal Medico-Psychological Association: Ninety-sixth Annual Meeting.—Irish Division.—Opening of Runwell Hospital.—Notices by the Honorary Librarian.—Notices by the Registrar.—Notices of Meetings.

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THE
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No. 347 [NEW SERIES No. 311.] NOVEMBER, 1937. VOL. LXXXIII

Part I.—Original Articles.

CONVULSION THERAPY IN SCHIZOPHRENIA.

By ALEXANDER KENNEDY, M.D., M.R.C.P., D.P.M.,
Assistant Medical Officer, West Park Hospital, Epsom; lately seconded to Psychiatric Research Unit, Maudsley Hospital.

THE following account is based on the writer's experience of convulsion treatment at centres abroad, on a review of the literature up to the present date, and on three cases recently treated at West Park Hospital, Epsom. It is published at this early stage in order to make a knowledge of the technique and literature of a method which has already proved itself worthy of extensive trial, available to a larger number of workers in this country.

Over a thousand cases have now been treated at various hospitals in Europe, and the mortality and remission-rates, especially in the catatonic type of case, compare favourably with the insulin shock treatment introduced by Sakel in Vienna in 1933 and developed in this country during the past year. The permanence of the remissions produced by this method cannot as yet be assessed.

HISTORICAL AND THEORETICAL.

In 1929 Nyiro and Jablonszky observed that cases of epilepsy in which schizophrenic features were present recovered rapidly when fits were frequent, and in 1930 Müller described two cases in which schizophrenic illnesses recovered rapidly after the appearance of spontaneous epileptic fits.

Glaus in 1931 pointed out that the combination of schizophrenia and epilepsy was a rare one, and of 6,000 schizophrenics investigated, he found only 8 who had had fits at any time of their life, and of these 4 recovered shortly after the fits appeared.

Although it is traditional in English mental hospitals that seizures of one sort or another are not infrequently found in chronic catatonic states, the writer has not been able on investigation to satisfy himself that true epileptiform convulsions have occurred in a single chronic catatonic at West Park Hospital, and in 8 cases in whose case-records seizures were reported, none had been recorded as epileptiform, and most of them seemed to have been of the nature of vaso-vagal syncope. Of 120 psychotic epileptics having frequent fits, only

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one showed a mental picture at all recognizable as catatonic, though paranoid features were, of course, common enough.

Straub, on examination of 6,000 cases of schizophrenia, similarly reports that though 20 cases were said to have had epileptic fits, he could not satisfy himself as to the diagnosis, or witness a fit in a single case. Conversation with experienced mental nurses tends to confirm the fact that when seizures occur in chronic schizophrenics, they are rarely of the true epileptiform type.

It would appear, then, that there is some antagonism between catatonic schizophrenia and epilepsy which does not allow them to occur together in anything like the number of cases that would be expected in view of the frequency of the two conditions in the general population.

It was a recognition of this antagonism that led von Meduna of Budapest, in 1934, following a series of experiments on animals, to produce epileptiform convulsions in the human subject by means of intramuscular injections of 25% camphor in olive oil. He showed that the readiness to react with a convulsion varied with the individual susceptibility, both in animals and in man, rather than with factors such as the body-weight. His first experiments in man were carried out on advanced deteriorated schizophrenics and produced little improvement in their condition, but when the method was applied to recent cases encouraging results began to be obtained at once.

The results, however, were very variable, and often severe side-effects in the form of delirium and prolonged vomiting due to the overdoses of camphor were encountered. The great bulk of the injections, their painfulness and uncertain effects led von Meduna to investigate other convulsant agents, and of these pentamethylenetetrazol has so far proved to be the most safe and reliable.

Chemical.

Pentamethylenetetrazol was synthesized by Prof. K. F. Schmidt of Heidelberg, during experiments directed at the synthesis of water-soluble substances having the therapeutic properties of camphor. His efforts met with no success until a discovery made by academic workers on the dissociation of hydrazoic acid revealed hitherto unknown reactions, by which it was possible to produce from ketones compounds of the type—

$$\begin{array}{l} R - C = N \diagdown \\ R_1 - N - N \diagup\!\!\!= N \end{array}$$

in which R and R_1 represent different organic groups of which the identity depends on the original ketone.

The basal ketone chosen by Schmidt was cyclohexanone because of its essential similarity to camphor but simpler structure:

$$\begin{array}{l} CH_2 - CH_2 - CH_2 \\ \quad | \qquad\qquad | \\ CH_2 - CH_2 - CO \end{array}$$

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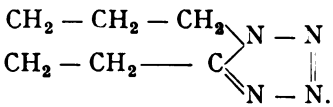
One of the resultant compounds was pentamethylenetetrazol:



This compound is of interest in that it represents the simplest member of a series of compounds not hitherto known and of very great theoretical importance, and it was fortunate that von Meduna was able to find in it a substance having the convulsant properties of camphor without its disadvantages of insolubility and toxic effects.

Pharmacology.

A very extensive literature has accumulated on the pharmacology of pentamethylenetetrazol, which is widely used in much smaller doses by cardiologists under the names of "cardiazol" and "metrazol" (U.S.A.). Its convulsive properties in the experimental animal have been known for a long time, but their application to treatment was not discovered until von Meduna's experiments. Some of the results of earlier work, however, have a bearing on the technique of convulsion treatment.

Although no very close similarity exists between the chemical formulæ of camphor and of pentamethylenetetrazol, experiments made by F. Hildebrandt showed that it had the stimulant effects of camphor with the advantages of solubility, ready absorption and elimination, and suitability for intravenous use. He found that in large doses convulsions were produced in rabbits resembling those produced by picrotoxin. Schoen showed that in smaller doses it increases the reflexes and antagonizes the action of sedative drugs of the sulphonal and barbitone groups—a fact that emphasizes the importance of avoiding these drugs during treatment if convulsions are to be obtained with minimal doses.

Experimentally, the irritability of the "vomiting centre" is increased (Schwartz), but fortunately in the smaller doses used clinically vomiting is not common, and the writer has not seen vomiting occur within an hour of a convulsion, though it has frequently been described.

It is also a powerful stimulant of the respiratory centre, and it is probable that this is the reason for the rarity of alarming asphyxial symptoms after the apnœic period, during the fit. Although there may be considerable cyanosis, if the airway is clear no anxiety need be felt.

The drug is a powerful stimulant to the vascular system generally, though in non-convulsant doses it does not greatly increase the blood-pressure. During a course of treatment, however, it is usual for the systolic blood-pressure between convulsions to rise day by day if recovery is going to occur. Although the large dose injected in this treatment causes the heart to beat very violently

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and the great vessels to pulsate, the pulse-rate is not greatly increased even during convulsions, and is much slower than during idiopathic epileptic fits.

Absorption and Excretion.

Owing to its ready solubility in practically all solvents, and in lipoids, pentamethylenetetrazol is rapidly absorbed, even if given intramuscularly, and about three times the intravenous dose when given in this way will usually produce a convulsion. On account of the variability of the time before the onset of the convulsion, and of the need to watch the patient for at least an hour after administration of an intramuscular dose, and owing to the greater uncertainty as to whether a convulsion will result the writer has not employed this form of treatment, though it is given by many continental workers, including von Meduna.

The final mode of elimination after injection is as yet uncertain. It is removed rapidly from the circulation after intravenous injection. Hildebrandt finds that the speed of detoxication of the serum in an average convulsive dose is 0·85 mg./kilo/minute, and other workers confirm this (Barker and Levine).

Biehler has shown that the destruction of the drug is so rapid that two doses of 1 c.c. of a solution, injected shortly after one another, do not produce in experimental animals anything like the effect of a 2-c.c. dose, and so rapid is this destruction that even the rate of injection affects the concentration of the drug in the circulation during subsequent minutes. This rapid disappearance is one reason for the great speed with which it is necessary to inject the solution. The other reason for injecting rapidly is to ensure that as large a quantity of the drug as possible can reach the vessels of the central nervous system at the same time, after it has traversed the heart, and before it has diffused itself into the general circulation.

It may be that excretion takes place, as suggested by Biehler, into the small intestine, but a large quantity must clearly be destroyed as it cannot be recovered unchanged in the excreta. Only small quantities can be found unchanged in the urine passed after a convulsion.

The rapidity of excretion or destruction is a great advantage in treatment, as the increased excitability of the central nervous system passes off very rapidly after the convulsion is over. As will be seen, it is necessary to produce a convulsion after each injection, and if additional solution has to be injected it must be done at once, and for this reason the needle is always left in the vein until the convulsion has actually started.

Physical and Chemical Properties.

Pentamethylenetetrazol is a white crystalline powder with a faintly bitter taste; it is soluble without decomposition in most organic solvents and in water, the aqueous solution being almost neutral. A series of solutions

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buffered to pH 8·0 remained at this pH after sterilization at 120° C. for half an hour, and showed no change after storage for a month.

Its presence may be detected by the production of a precipitate on addition of a saturated solution of mercuric chloride to a solution, and this is sensitive up to ·0001%. The reaction, however, is not specific, as it is given by a number of reducing substances and by some alkaloids, including quinine.

The solution gives Zwicker's reaction, i.e., if potassium bichromate be added to an acidified solution and shaken with hydrogen peroxide a blue colour is produced, which dissolves in chloroform when the latter is shaken with the solution. The test is sensitive to ·0005%, but can be made more sensitive by the addition of an acid solution of cuprous chloride, which makes it sensitive to ·000001%. By means of these tests it was possible to detect small quantities of the drug in the urine of patients during the first hour after convulsions. It can usually be so found when a convulsion has been successfully produced and indicates that the speed of injection has been high.

The solution therefore is stable and soluble. To some extent it inhibits the growth of bacteria; v. Angerer showed that 1% and 10% solutions in saline had mild bactericidal properties. The writer, however, has found that at least 5% must be added to a broth culture to inhibit the growth of staphylococci and coliform bacilli.

Phenomena of the Convulsion.

The usual interval between the commencement of the injection and the first sign of the convulsion is about 10 seconds. If the first signs are delayed for more than 20 seconds, the ensuing convulsions are likely to be incomplete and unsatisfactory. The fit usually comes on with great suddenness only a few seconds after the premonitory signs. These are sudden pallor, a very characteristic short cough, twitchings of the fingers and movements of the facial muscles, which give the patient the appearance of being suddenly terrified. These signs are not usually all present together, but one or more can always be seen.

Very often the terrified expression is followed by a few staring glances from side to side, dilatation of the pupils, and a cry or scream. The staring eyes then cease to move, and slowly turn upwards or deviate to one side as consciousness is lost.

The tonic state usually commences with a few twitches in the hands or feet, the hands then going into the tetanic or "accoucheur" position, or the feet plantar-flexing while the spasm rapidly spreads all over the body. Frequently one side is affected a moment before the other.

At the onset of the tonic phase in most cases the mouth opens widely, and if this moment is watched for a gag or airway can be inserted before the jaw closes.

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This sudden opening of the mouth appears to be a peculiarity of the artificially induced convulsion, and accounts for the frequency of dislocation of the jaw as a complication. This occurred in one of the writer's cases.

The whole body then goes into violent opisthotonos, with the muscles quivering and tense, the face gradually becoming flushed, and the veins standing out on the neck. Respiration ceases and the arteries in the neck can be seen pulsating violently, the rate being usually about 90—rarely over 100.

The tonic state lasts from 10–30 seconds, this time varying, according to von Meduna, with the individual and not with the dosage. Other workers, however, have found the tonic state to be prolonged if more than the necessary dose be given.

The clonic stage is ushered in by short periods of relaxation, which increase in length until clonic convulsions are present. This stage lasts from 15 seconds to a minute and during it the muscles are irritable, although Chvostek's sign is absent, and the reflexes are increased and one or both plantar responses is extensor. The pupils are dilated, but may quickly contract at intervals. The pulse usually becomes more rapid as cyanosis begins to appear but rarely exceeds 120. No change can be seen in the retinal vessels at any stage of the convulsion, except possibly some widening of the veins when cyanosis is marked.

As the clonic spasms become less frequent and there are no respiratory movements, cyanosis increases and may become marked. If, however, the airway is clear, there need be no anxiety. There is sometimes an interval before respiration starts again, but if this is too prolonged a single pressure on the chest-wall with the open hand will usually start it off. The cyanosis is rarely as deep as that seen in a badly administered N_2O anæsthetic.

After the clonic stage proper, a few single convulsions may occur during the next minute and the muscles may remain irritable. If the shoulder-girdle muscles are put on the stretch by pulling on the arm, for about a minute after the clonic phase they will contract, but after this sign has gone the patient will lie still. The cough reflex soon returns, and if an airway has been put in, movements will be seen in the patient's throat.

Patients frequently micturate during the fit, and in the recovery stages they are often distressed to find that this has occurred.

Unconsciousness lasts for a variable period ; in most cases 5–15 minutes, and is recovered from gradually, the patient often showing momentary signs of fear resembling those at the onset of the fit. He is invariably disorientated at first and is unable to recognize those around him, but if his contact with his environment before the convulsion has been good, he will regain his orientation in a few minutes.

The patient is usually unwilling to speak and has to be pressed to answer questions at this stage. After a few minutes of restless movements and

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attempts to get out of bed he will usually curl up and go to sleep. During this short restless phase, which does not occur in all cases, the patient may wander about in an aimless fashion, and must not be let out of sight for more than a few moments.

There is no transient remission in the illness corresponding with that seen after insulin therapy, or after the administration of intravenous sodium amytal, and the patient returns after the convulsion to approximately the same state as before its induction.

After 1–2 hours' sleep the patient usually wants to get up, and will often say that he has had no food or that he is remarkably hungry ; in fact, throughout the treatment appetite is always very good and tube-feeding is rarely necessary after the second convulsion, even in cases that do not recover. This observation has been made by most workers, and it has a practical application in that by a few injections cases of chronic catatonic stupor may be made to feed themselves even if they are unlikely, on account of their chronicity, to have remissions.

There appears to be no objection to patients getting up 2–3 hours after the convulsion. They will usually state that they are tired, but as a rule have surprisingly little comment to make even when recovery is practically complete.

In the majority of cases there is a complete amnesia for the period of the fit and for any abnormal behaviour which has occurred during succeeding minutes. Thus a patient who tried to climb out of the window 10 minutes after the fit went to sleep on being put to bed, and could recall nothing of the incident on the following day.

Metabolism during Convulsion Treatment.

A number of investigations have been done which allow of some comparison between metabolism in induced and in idiopathic convulsions. During the three-hour period following a convulsion there is a decrease in the urinary acidity, a reduction in the ammonia content, a slight increase in the phosphate content and a considerable reduction in the urinary chloride.

These findings are similar to those found in idiopathic epilepsy.

Effect of Non-Convulsant Doses.

Doses of 1 c.c. of the solution used in this treatment produced little in the way of subjective phenomena apart from palpitations and sometimes vertigo, but as the dose given approaches the convulsant level (4–12 c.c.), as when the earlier doses are being given in a course of treatment, the injection causes a disturbance of the patient's mental state which may last for several hours.

In mild or recovering cases this often takes the form of a severe anxiety-attack in which the patient sits up in a frightened manner, may ask those standing by not to come too near, and may give nervous glances about him as

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if in fear of some unseen danger. Consciousness is not lost in these cases and the patient remembers what has happened in a vague way, and the memory is an unpleasant one, which may make him hesitate to have further injections.

In other cases, especially early in treatment, the patient may go into a state of catatonic excitement for several hours, showing inconsequent activity, being alternately erotic and aggressive, smashing crockery, stripping off his clothes, etc. This state has generally cleared up in 5 or 6 hours.

In view of the unpleasantness of non-convulsant doses, it is considered that once the dosage for the individual patient has been established, it is essential that a convulsion be successfully induced after each injection. When there is no fit the mental disturbance seems to last longer, whereas when a fit has occurred the patient usually returns to his previous state within 3 hours at the most of the injection.

Technique.

General Purpose of the Method.

The object of the technique is to give the patient a series of epileptiform convulsions without harming him in any way, and this is achieved by giving intravenous injections of the solution to be described, in such a manner that as large a quantity of the drug as possible reaches the vessels of the central nervous system at the same moment. A series of such convulsions are given until a remission is obtained, or until it is manifest that there is no improvement.

The case selected must, of course, be a suitable one according to the criteria of case-selection to be mentioned later.

The patient is put to bed for two days and the pulse, temperature and respiration are taken 4-hourly during that period. A general physical examination is done, and, if the patient will co-operate, an exercise-tolerance test. The urine is examined on two consecutive days, and then weekly throughout treatment. The Wassermann, blood bromide and blood urea are determined and a blood-count is done if necessary. As most of the patients are stuporose, tube-feeding is often necessary, and if enough food is not being taken this should be resorted to without hesitation in order to get the patient into the best possible condition before the treatment is commenced. The blood-pressure is taken and recorded on the patient's chart with the data above-mentioned.

A diagram is next made of the veins in each antecubital fossa, so that the site of each injection can be recorded and the presence of inflammation, hæmatoma; or thrombosis marked in.

If any of the solution has been injected outside a vein, or if there is any bruising, that vein is, if possible, left alone for ten days. If thrombosis occurs, tributary veins of the thrombosed vein are avoided, in order to prevent

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mobilizing the clot. The above precautions are necessary on account of two factors; firstly, the tendency of the solution, if allowed to be in contact with a vein for long, to cause thrombosis, and secondly, the high pressure at which the solution must be injected, as this tends to cause leakage if the lumen of the needle is not completely in the vein, with consequent paravenous inflammation and risk of thrombosis. These difficulties have been encountered by all the workers seen by the writer, and appear to be almost unavoidable in some cases. The difficulties arise in the great necessity of causing a maximum concentration of the drug to reach the central nervous system at the same moment.

Method of Preparation of the Solution.

The writer has used the 10% solution throughout his experiments, as weaker solutions made it necessary to inject inconveniently large quantities, or at an impracticably high rate. Stronger solutions have been universally abandoned since the Berne-Münsingen Congress, where several cases in which unpleasant sequelæ had occurred were described, and in addition it has been found that if this solution is not injected accurately into the vein necrosis of the surrounding tissues may follow and thrombosis of the vein is very frequent.

In order to reduce the tendency to local reactions the solution has been buffered to pH 8 by the addition of 0·1% of disodium hydrogen phosphate (Na_2HPO_4, $12H_2O$), the formula used being:

Pentamethylenetetrazol . . .	10 grm.
$Na_2HPO_4 12H_2O$	0·1 ,,
Redistilled water to	100 ml.

This solution is immediately transferred to rubber-capped 25 c.c. vaccine bottles made of resistive glass, soda glass being unsuitable as the pH is apt to be altered during autoclaving. The bottles are then sterilized by autoclave at 120° C. and 15 lb. to the square inch for half an hour. After sterilization samples are tested for pH as a precautionary measure, but these were never found to vary appreciably, indicating that no decomposition had taken place. A number of 5 c.c. ampoules were also supplied by Messrs. Knoll A.-G. and these were found very convenient, but some of the solution is apt to be wasted when ampoules are used, as the dose varies from time to time. As the solution is mildly bactericidal there appears to be no objection to the use of vaccine bottles provided careful aseptic precautions are taken, and their use effects a considerable reduction in cost.

Method of Giving the Injections.

When the injections are given to a group of patients at the same time, it is essential that no one patient shall see another having treatment, and for

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this reason the injections were given in small rooms off a main corridor, all the cases being put to bed before the first injection was given.

It was also found advisable for patients having the treatment not to meet one another, especially in the latter stages, as in this way it was often possible to conceal from the patients that they had had convulsions, and to speak to them of the results of the injections as " a period of unconsciousness ", which was useful at the stage of recovery, when most patients resented further treatment.

The injections are best given in the morning, no food having been taken except 4 oz. of glucose half-an-hour before. It is necessary to withhold all sedatives with the exception of paraldehyde and hyoscine, as when others are given it is at once necessary to give larger doses of the solution in order to produce convulsions. The writer has found that hyoscine has no inhibiting effect on the convulsions ; there seems to be no objection to giving the treatment to patients premedicated with this drug, and this may prove to be a valuable method in the treatment of resistive patients.

The patient it put on a low bed or mattress in the centre of a room, the head being at the lower end of the bed so that the bed-end does not interfere with manipulations. The head is slightly raised by means of a pillow and the whole bed is covered with a mackintosh sheet and blanket. The mouth is examined for dentures.

It is absolutely essential that the needle shall not move once it is in the vein, and as it has to be left in the vein until the fit has actually commenced, the arm must be held very firmly in full extension. The patient is not held in any way, however, until immediately before the injection, as this would add to the apprehension which patients feel before the injections, in the later stages of recovery.

The syringe used is a 20 c.c. Record with an eccentric nozzle and the needle is of $\frac{8}{10}$ bore with a short bevel. Larger $\frac{11}{10}$ needles were used at first as this allowed of rapid injection without using any force, but it was found that veins were damaged more often with this size. The solution, drawn from rubber-capped bottles or ampoules, is put into the syringe before the room is entered, so that the patient does not see the syringe for more than a few seconds. 3 c.c. more than the determined dose are put into the syringe. The tourniquet used to distend the veins is a soft rubber tube, as this can be taken off more quickly than a pneumatic armlet or band and clamp.

As soon as the physician enters the room the arm is straightened out and the antecubital space painted with spirit, and the needle put into the previously selected vein. After blood has been withdrawn into the syringe the needle is pushed, if possible, about 2 cm. up the vein, so that there can be no chance of leakage at the point of entry due to the high pressure at which the solution is injected. The tourniquet is then slackened by pulling on the rubber tube, and the solution is injected as rapidly as possible up to the determined dose. The

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remaining 3 c.c. are held in the syringe, and if no fit occurs within 20 seconds this is also injected.

As soon as definite premonitory signs of the convulsion appear, the needle is withdrawn as rapidly as possible. There is little doubt that if the injections are given very quickly, less of the solution is required.

The importance of obtaining a full convulsion with each injection cannot be over-estimated, as if none is obtained the patient is usually anxious and unsettled for several hours, and invariably has unpleasant memories of the event which make him less inclined to co-operate.

The assistant in charge of the mouth and the gag kneels at the head of the bed with her hands on either side of the patient's head. When she has heard the characteristic premonitory cough she waits for the opening of the mouth which usually indicates the onset of the generalized tonic convulsions, and tries to prevent the mouth from opening too wide in order to prevent dislocation of the jaw. As soon as the first shock is over she puts into the mouth a gag made up of three plywood throat spatulas bound round with bandage. This prevents the mouth from closing and obstructing respiration. After the eyes have turned up and consciousness is clearly lost a Hewitt's airway may be inserted, and this has the advantages of preventing the tongue from falling back. The airway must be removed as soon as the cough reflex returns, usually very soon after the clonic stage is over.

It is not usually necessary to restrain the patient during the convulsion, and it is best allowed to take its course unless the patient would otherwise fall off the bed. Unless the patient is in a state of excitement and actively resists treatment a single assistant may be sufficient, though two are preferable, as one can devote her attention to the arm and tourniquet and the other to the jaw and gag.

It is necessary that one nurse remain with the patient for 10 minutes after the convulsion, after which a single nurse may watch two or three patients by walking from one room to another along the corridor. In this way three or more patients may be treated with ease with three nurses, one being left behind to watch the patients after the injections are over.

On awaking from the sleep which usually follows a convulsion, patients are hungry and are allowed to eat a full lunch in bed, and to get up soon after. If co-operative, they usually soon express themselves tired and go to bed early and sleep well without a sedative. Should a sedative be required, paraldehyde is the only one permissible owing to the antagonism of most drugs to the convulsant agent.

The interval between injections varies in different clinics from 2 days to a week. When, in the recovery stages, the patient shows an alteration in mental state after each injection, e.g., excitement, this alteration passes off within 48 hours, when the patients resume their former state, and for this reason the writer gives another injection after that period, i.e., on alternate days.

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After remission has occurred it is usual to give three further injections at bi-weekly, and then at weekly intervals in order to prevent relapse.

Dosage.

The dosage necessary to produce a convulsion varies from 4–16 c.c. of the 10% solution. The amount necessary to produce a fit is of considerable prognostic value, as cases in which it is necessary to give more than 10 c.c. rarely remit. With the cases described in this paper it has never been necessary to give a greater single dose than 9 c.c.

The correct dose is found by starting with an injection of 5 c.c. and giving 1 c.c. more each day until the convulsion is produced. The reason for this rapid increase is that if many injections are given without producing a convulsion, anxiety attacks and an unsettled state which is very unpleasant to the patient, may occur. Once a convulsion has been produced, it is essential that another occurs with each injection. For this reason 3 c.c. more than the determined dose are taken into the syringe so that more can be injected if there is no convulsion. The rapidity of excretion is such that this can be done with safety.

Once the patient has started on regular injections every other day it is often possible to reduce the dose by giving ·5 c.c. less on each occasion until it becomes necessary to inject the 3 c.c. kept in reserve in the syringe. A chart is kept showing each dose given and its effects.

Effects on the Mental State.

The effects on the patient's behaviour may be divided into the immediate effects brought about by each convulsion, and the total effect brought about by a series of convulsions.

The phenomena immediately following the convulsion have already been described. The patient rarely remembers much of what has happened in the period immediately after the fit. For the rest of the day in stuporose patients there is little change, except that there is usually no difficulty in getting them to take nourishment, even when they have been tube-fed before, and that the extremities, if they have been cyanosed before, are generally quite pink, and œdema rapidly begins to disappear.

Patients who can co-operate say that they are tired and sometimes complain of backache, and are usually glad to go to bed early. On the following morning the patient is almost invariably more active than before, and many of them, even those who have been in stupor, become excited and may refuse to wait to get up at the normal time and will dash out of their beds and run about, often stripping off their clothes. When they have got up they may shout, sing

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and quarrel with other patients and quite often make foolish attempts at suicide or wantonly attempt to smash crockery, so that they have to be confined to bed, as they cannot otherwise be controlled. Their conversation at this stage is irrational and they show gross thought-disorder. They are very frequently hallucinated, usually aurally, but sometimes visually, and may be careless or even degraded in their habits. By the next morning the patient has usually returned to his former state, or is somewhat improved. Throughout treatment appetite is invariably good and the bowels usually act several times during the day, though severe diarrhœa is not recorded.

Owing to this tendency to overactivity, it is advisable to find occupation for the patients as soon as they are sufficiently recovered to permit of it, and they often work well on the days on which they are not having their treatment.

In more co-operative cases, a conspicuous feature is the lack of curiosity about the treatment in the earlier stages. This is later frequently replaced, as the thought-disorder improves, by a violent antagonism to it, which is, however, rarely enough to make the patient fight against the injections in a really purposeful manner, though he may refuse at first.

The effects on the mental illness itself brought about by the convulsions take place gradually, as a rule, and transient remissions such as are seen in insulin treatment and after intravenous sodium amytal are not found. von Meduna describes three types of remission : *rapid*, taking place early in the treatment and remaining permanent ; *progressive*, by far the commonest type ; and *intermittent*, with a series of remissions of increasing length.

Most workers have commented on the uniformity of the changes in the mental state in a great many cases, and especially on a euphoric state which often appears before recovery.

Many patients pass through several phases of schizophrenic disorder during the treatment, a catatonic stupor, for instance, developing excitement and then paranoid ideas with hallucinations, finally becoming euphoric and friendly as the remission comes on, passing in a few days through phases which might take months or years if they occurred at the usual speed of development of a schizophrenic illness.

As will be seen, two of the writer's three catatonic cases passed first into a state of excitement, in which they were co-operative and over-active, then into an anxious phase in which they seemed to dread some catastrophe and were hallucinated for hearing, and then into a euphoric-erotic state in which they welcomed the treatment unmistakably as some sort of sexual experience. Following this they thought more clearly, discussed the treatment with one another and decided that it was unreasonable, though at this time both admitted that they felt far better and attributed their improvement to the injections, and both refused further treatment, though they could always

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be persuaded to take it at the last moment. The third patient changed in exactly the same way up to the euphoric-erotic stage, when treatment was abandoned ; she then relapsed completely within a few days.

It is premature to form an opinion on the permanence of the remissions produced. Many of the earlier cases have relapsed and had second courses of treatment ; others have unexpectedly remained well, as often occurs after spontaneous remissions in schizophrenia. The number of relapses in some clinics has considerably shaken the optimism with which the method was first undertaken. Even if the very high rate of recovery at first claimed is not maintained, however, the number of relapses is not sufficient to bring this rate as low as the spontaneous recovery-rate.

Results of the Treatment.

Well over 1,000 cases have now been treated under well-controlled conditions by von Meduna, Angyal and Gyarfas, Klaesi, Georgi, Wahlmann, Lehmann-Facius and others, and most of this material was at the disposal of the recent Berne-Münsingen Congress. The main conclusions were :

1. Only early cases are improved. The remission-rate falls rapidly with the duration of the illness and practically no cases of over four years' duration have recovered.

2. The catatonic form makes the best response, especially stuporose cases.

3. With selected cases a complete remission-rate of over 70% can be obtained, not including partial or " social " remissions.

Selection of Cases for Treatment: Prognosis.

The original rules for case-selection laid down by von Meduna have stood the test of further investigation. He stated in his first paper that favourable results might be anticipated in early cases, especially if the onset of the illness is sudden and not developed gradually from an eccentric personality or superimposed on mental defect, and in cases where there is no serious psychopathic heredity. He says that remission occurs only when the individual is constitutionally able to react favourably to the treatment, and assesses this ability to react by the patient's readiness to respond with convulsions to small doses. He has also introduced a hæmatological test of his own, namely the measurement of the extent of the shift to the left of the Arneth and Schilling counts on the leucocytes during the three hours following a convulsion. There is little doubt that the response to a small dose is a good prognostic sign, but the value of the hæmatological test is still in doubt, as the shift is usually of slight extent and does not seem to be specific for induced convulsions.

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Discussion as to the type of schizophrenia which reacts most favourably to the treatment has been ambiguous owing to the differing ways of describing clinical syndromes, but there is little doubt that on the classification of Kraepelin the catatonic form reacts best and the paranoid is least affected, in contrast to the results of insulin treatment.

Mortality.

The literature available contains reference to four fatalities during treatment. In two of these, gross disorders that had clearly been present before the treatment began were found at autopsy—aortic regurgitation and hypernephroma. The other two deaths were due to pulmonary embolism, and in view of the tendency to thrombosis of the veins it would appear that these deaths could be attributed to the treatment. If, however, precautions are taken against the formation of, and mobilization of clots, such as are described in this paper, the incidence of pulmonary embolism should be very small indeed—certainly less than that encountered after routine surgical operations.

Comparison with Insulin Shock Treatment.

Although shock treatments for schizophrenia of various kinds, endocrine, pyretic and chemical, have appeared at intervals during the last twenty years and have failed to prove their value with large series of cases, or have proved to be of only limited application, it is remarkable that two widely differing methods in insulin shock and pentamethylenetetrazol convulsion treatment should have appeared within a year, and at once commanded attention on account of their high remission-rates in comparatively large series.

The methods are different in many respects, both in the type of case affected and the mental changes following their administration, and even the convulsions which they have in common probably differ in their origin, those following insulin being of the nature of negative release-phenomena, while those induced by pentamethylenetetrazol appear rather to represent a positive cerebral disturbance.

A few workers have studied the two methods in detail side by side, notably Angyal and Gyarfas of Budapest and Klaesi of Berne. The former have attempted to judge the remissions brought about by the two methods by the same standards, and are able to demonstrate that the type of case most benefited by each of the treatments is not the same, the stuporose cases being little affected by insulin but most influenced by convulsion treatment, while the reverse is the case with early paranoid forms.

Their most interesting observation in this connection was that in a small series of early cases treated with convulsions, a remission-rate of 44% was secured, counting only complete remissions, but when the unsuccessful cases

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were submitted to insulin therapy 22% more remitted, giving a total remission-rate of 66%. This further supports the view that the two treatments act on different types of case.

The two methods, therefore, are probably mutually adjuvant in the treatment of schizophrenia as a whole, and choice between them will probably depend in the future on the clinical features of the cases, rather than on the relative ease of handling of the methods themselves.

Statistically, convulsion therapy would appear to be the safer method, the general mortality on the cases described at the Berne-Münsingen Congress being about 0·3, whereas in Küpper's series of insulin cases it was 1·5%, though, as James points out, this was at a time when the technique had yet to be improved.

In the case of insulin therapy the patient must be under close observation for a much longer period—6 hours at least—than with convulsion treatment, and the possibility of emergencies arising is much greater; in fact, serious emergencies requiring intracardiac injections, infusions and the administration of stimulants are of frequent occurrence, whereas, even with the rigid technique outlined above, the active period of the convulsion treatment is very brief.

The writer has recently had an excellent opportunity of comparing the difficulties of the two methods by seeing, on the same day, the carefully controlled insulin technique under Prof. Schneider at Heidelberg, where the physician never leaves his patients throughout the day, and has to have every means of restoration ready for the very frequent emergencies which occur, and the convulsion therapy under Dr. Lehmann-Facius at Frankfurt, where a similar number of cases were treated without incident within the space of little more than an hour.

Both methods, however, are both spectacular and dangerous, and unless a rigid technique is maintained avoidable complications are bound to occur even in the simpler convulsion treatment, and the haphazard treatment of unsuitable subjects under insufficient control can only lead to misfortune.

The idea, originated by Klaesi, that the treatments should be combined, and convulsions induced in the course of insulin coma, has received a good deal of support, but at the Berne-Münsingen Congress the general opinion was that this should be avoided on the grounds that enough is not yet known about the methods individually, and that such combination would tend to make an understanding of their mode of action more difficult.

The principal advantage of the combined treatment is that the convulsions can be induced while the patient is unconscious, and the unpleasantness which the non-amnesic patient suffers is avoided. This would appear to be the only justification for combining the treatments at the moment, but the giving of insulin and pentamethylenetetrazol on different days, as practised by James, savours of comprehensive therapy, and must lead to doubt as to which was the successful therapeutic agent, if remission occurs.

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Case-Material.

Case 1.—E. R. E—, aged 23; single; domestic servant. Admitted as a temporary patient in a deep catatonic stupor in which she had been for about a fortnight. Remained stuporose for nine months before convulsion treatment was commenced.

Family history.—No gross psychoses among near relatives except that her mother had a menopausal depressive attack lasting three months. A healthy country family, the last generation of which had migrated to London.

Previous personality.—Always a rather quiet girl; no men friends except a tentative affair a few months before the onset of the illness. Worked well and used her leisure in a normal manner, and was not solitary. No previous breakdowns.

Present illness.—During the six months preceding the onset of her illness she had become inclined to complain, had frequently grumbled to the housekeeper about the quality of the food and had become discontented with her work; had spoken about taking up nursing, and had made arrangements to start working in a fever hospital. Two months before her admission to the observation ward, she told her sister that she felt that people would get to know that she had grumbled about the food, and that this would hinder her nursing career.

She also began to eat very little and to be grossly over-conscientious over her work, and a month later said that she had pains in her head caused by the housekeeper shouting abuse at her during the night. After this hallucinations became more prominent and she began to hear her father speaking to her, and she then, quite suddenly, went into a negativistic stuporose state and was admitted to the observation ward; being regarded as non-volitional, she was sent to West Park Hospital as a temporary patient.

State on admission.—In a deep, mildly negativistic catatonic stupor, requiring continuous hand- and occasional tube-feeding. Completely mute and incontinent of urine and fæces. Extremities cyanosed and feet swollen. No abnormal physical state apart from evidence of old tuberculosis at one apex corresponding to a history of having been treated for this when aged 16.

Progress up to commencement of treatment.—Remained in stupor for nine months, requiring every attention. Never spoke a sentence or co-operated in any way and gradually became more resistive.

Physical state at commencement of treatment.—Extremities very cyanosed. Blood-pressure 105/60. Poor air-entry at left lung-apex but no signs of active disease in X-ray. Heart-sounds normal. Exercise-tolerance test impossible on account of mental state. Urine normal. Wassermann reaction negative.

Details of treatment.—Twenty-four convulsions in all. Average dose 5 c.c. of the solution. Two injections were given without producing convulsions and an anxiety attack developed in each case. Complete amnesia for all convulsions. Last three were given at 4-day intervals, after remission was complete; others on alternate days. Total time, 8 weeks.

Progress during treatment.—After the second convulsion, spoke a little and began to feed herself. Œdema of ankles disappeared and veins in the arms became more prominent, making injection much easier. Blood-pressure 106/68.

After the fifth convulsion she became excited and said she was not going

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to allow further injections. Was allowed out with her sister, who reported that she was able to converse on general topics.

After the eighth injection she became impulsive and remained so for a week, wandering about aimlessly and saying that something dreadful was going to happen.

After this she developed a paranoid trend, and for a week said that the injections were being given to hurt her heart and chest, and that these had not been properly examined. Blood-pressure 111/68. She then, five weeks after commencing treatment, began to become euphoric and rather erotic, saying that the treatment was giving her new strength, and holding out her arm asking for more injections. At this point she became volitional for legal purposes, and was able to say that she was ill, where she was, etc.

From this time onwards she developed increasing insight; was able to do useful needlework, and gradually lost all evidence of schizophrenic thought-disorder.

At the conclusion of the treatment she was made a voluntary patient and decided to stay for a short convalescent period before returning to work.

Patient's retrospective account.—She was able to remember the period of her stupor, giving the usual account of hearing everything and being unable and disinclined to do anything. She is quite unaware that she has had any fits, but knows that the injections made her unconscious. "It was always the same: I heard a buzzing in my head and felt my right arm come up. I would turn over and feel I was going to say something, and I could feel my mouth opening—and then I would wake up suddenly and find everybody gone, except the nurse at the door." She was therefore amnesic for the short period of anxiety before the fit, for the fit itself, and for the period after the fit when she used to try and get out of bed, and would answer questions put to her. She thus appeared to recover consciousness long before she actually did so.

Her blood-pressure at the conclusion was 122/74.

The above case is the only one which was eligible for treatment by von Meduna's criteria for case-selection. Her prognosis by the usual rules was probably quite good, though her illness had lasted nearly a year. Recovery was complete and she remains well.

The two cases which follow are still under treatment; they are of longer standing than the previous one and would not be considered to have a good chance of recovery according to von Meduna. Nevertheless, the first case made a considerable improvement, but relapsed completely when treatment was abandoned owing to difficulties in injecting the solution.

CASE 2.—D. L. P—, age 21, female; single; no fixed occupation. Admitted to St. Ebba's Hospital twenty months before the commencement of treatment as a case of catatonic excitement and transferred to West Park Hospital under certificate one month before.

On admission was in a mild stupor alternating with outbursts of excitement.

Family history.—This was unsatisfactory as the patient had run away from home, but several members of the family were described as unstable.

Previous personality and illness.—Was a "model child", but later was

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often worried over trifles and was regarded as "hysterical", excitable and self-willed.

Five years ago had "nervous debility", and was in a mental hospital for four months in what appears to have been a catatonic state. Since then had been unable to keep in employment, and at the onset of her present illness was in a Salvation Army hostel.

Present illness.—Nothing known of the onset except that she was observed at the hostel to have foolish mannerisms, and is said to have made an attempt at suicide. At St. Ebba's Hospital she was alternately stuporose and excited, and in the latter state she was noisy and violent and had frequently to be secluded.

State on admission.—Unable to give a satisfactory account of herself. Often negativistic and impulsive. Showed marked thought disorder and often spoke unintelligible gibberish, and was evidently hallucinated for hearing. Incontinent of urine and fæces.

Progress.—Remained inco-operative and unemployable, with frequent outbursts of excitement. Was treated for pyelitis.

Physical state at commencement of treatment.—Heart and lungs normal. Blood urea and bromide within normal limits. Wassermann reaction negative. Urine shows a few pus cells and some albumin. Has intermittent glycosuria.

Details of treatment.—Eight convulsions on alternate days. Treatment then abandoned owing to difficulty in giving the injections.

Progress during treatment.—After the first two convulsions she became very excited, and could only with difficulty be kept in bed and attempted to tear up her clothing and smash crockery.

After the fifth convulsion she became euphoric, offered to do some work and made herself very useful, and after this improved steadily for a few days and was able to discuss her case. It was ascertained that she had no memory for the convulsions. At this point it became impossible to give any more injections, and within a week she relapsed completely and has shown no improvement since. Treatment has now been restarted.

CASE 3.—V. C. T—, aged 24, female; single; domestic servant. Admitted three months before treatment was commenced in a mildly stuporose state, having been confused and at times impulsive since the birth of an illegitimate child four months before.

Family history.—Two siblings are schizophrenic. Parents have lost all their money, and have come to live in an untidy flat without employment through sheer inefficiency. They deny all mental troubles in the family, but four out of five members of the family seen by the social worker have been unemployables.

Previous personality.—No previous mental illness, but has given great anxiety to her family and friends through difficulty in keeping employment, waywardness and alcoholism.

Present illness.—Sudden onset four months before commencement of treatment following the birth of an illegitimate child. During pregnancy, however, she thought that neighbours were talking about her. Three weeks after the birth of her baby she ceased to speak and took no interest in the child or in her surroundings. She sometimes became depressed and said the baby had died. She finally attempted suicide by cutting her wrist and was sent to an observation ward.

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State on admission.—Lies in bed with the bed-clothes over her head and shows no interest in her environment. Smiles and giggles at intervals and will make only monosyllabic replies to questions. Refuses to eat and requires hand-feeding. Hallucinated for hearing. At times dashes suddenly out of bed without reason. Frequently incontinent of urine.

Progress.—Gradually became less accessible and more stuporose during the three months before treatment.

Physical state at commencement of treatment.—Heart and lungs normal. Wassermann reaction and blood bromide normal. No anæmia; urine normal; blood-pressure 108/70.

Details of treatment.—Twenty-nine convulsions to date. Average dose 5 c.c., given on alternate days. On two occasions on which no convulsion occurred she had very severe anxiety attacks and was unsettled for the whole day. Time taken up to the present, 11 weeks. Dislocated jaw on several occasions, but this was easily reduced.

Progress during treatment.—After the first few injections she became very excited, running out of bed and at times saying that people were going to kill her.

After 10 injections her excitement took on a euphoric tone and she announced that the injections were going to make her live for ever. There was much erotic behaviour, and she referred to the injection as if it were a sexual experience.

After 16 injections she began to work and wrote home for pocket-money and was able to spend it sensibly. She still has impulsive outbursts. Blood-pressure 112/70.

During the next two weeks she became quieter for much longer periods and was able to do sewing and write letters.

Since then her condition has fluctuated considerably and she still shows thought-disorder, though she is able to give a fair account of herself. The treatment is being continued.

Patient's retrospective account.—She is unaware that she has had any convulsions. She appears to have a very unpleasant feeling as she becomes unconscious: "I can hear myself holler out and I feel awful, as it I were sinking down and was not ever going to wake up again, but I feel all right when I wake up." She apparently recovered consciousness rapidly, but was amnesic for the period in which she appeared to be gradually orientating herself, and had no memory for the questions asked of her.

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AN UNUSUAL COMPLICATION OF INSULIN SHOCK (HYPOGLYCÆMIC) THERAPY.*

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INTRODUCTION.

SALM (1) has recently described stuporous conditions which developed in schizophrenic patients undergoing the insulin shock treatment. He concludes that such cases constitute a serious problem, because, if they do not yield to the repeated administration of sugar, one is at a loss in regard to specific treatment.

Recently, while giving this insulin shock treatment to six patients, we were surprised to find that one patient on the twenty-second day of treatment did not awake from the hypoglycæmic coma on being given food. Intravenous glucose and adrenaline also failed to revive her. Blood and spinal fluid sugars were then found to be excessive, and anti-diabetic treatment (including insulin) was administered with success.

PART I.

The patient a female, aged 36 years, was admitted to hospital on April 25, 1935.

The duration of the mental illness was 11 years. Treatment was begun on April 6, 1937. Patient, fasting, received 10 units insulin in the morning, and, a few hours later, received food. The amount of insulin was increased daily by about 5 units. For 21 days there was no major incident. Detailed notes were made each day. It will be sufficient to give the notes of one or two days during this period:

Second Day. April 7, 1937.

(1) Time and amount of insulin: 10.30 a.m., 15 units.
(2) Time of food: 1.30 p.m.
(3) Blood sugar: 10.25 a.m., 65 mgrm. per 100 c.c.
(4) *Clinical notes.*—No mental change; diarrhœa during the afternoon.

* A communication read at the Quarterly Meeting of the Irish Division, Royal Medico-Psychological Association, held at Kilkenny Mental Hospital, July 24, 1937.

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Fourth Day. April 9, 1937.

(1) 10 a.m.: 25 units.
(2) 1 p.m.: Food given to terminate day's treatment.
(3) 11.50 a.m.: Blood sugar, 45 mgrm. per 100 c.c.
(4) *Clinical notes.*—Had an excited episode at 11 a.m., when she became restless, got out of bed and made it impossible to take blood for blood sugar estimation, which, however, was taken at 11.15 a.m., when she lay again quietly in bed. At 1 p.m. she was perspiring profusely; while taking her meal at this hour she was noticed to be drowsy, to doze off in such a way as to hinder the taking of food, so glucose D (1½ oz.) was administered in a glass of water at 1.10 p.m., and she quickly revived. Heart action good.

Fourteenth Day. April 19, 1937.

Prominal as usual (3 gr. had been given for some days previously, before the insulin injection, on account of a tendency to develop epileptic seizures during hypoglycæmia).
(1) 8.30 a.m.: 40 units insulin.
(2) 12.50 p.m.: Food given (glucose per nasal tube).
(3) 12.35 p.m.: Blood sugar 35 mgrm. per 100 c.c.
(4) *Clinical notes.*—10.15 a.m.: Perspiring; beginning to be drowsy; had much more peaceful coma than on previous days until 12.15 p.m. when she suddenly seemed to wake up and rolled around in the bed. Soon she dozed off again, but resisted a good deal and jerked herself all over the bed and gave a good deal of trouble in the taking of blood, at 12.35 p.m. She was like this—in a kind of "coma vigil"—until 12.50 p.m., when she was fed nasally with glucose. She then had shivering and tremors while awaking. She seemed more accessible for some time afterwards. She answered questions apparently quite normally, but without much initiative.

From April 6 to April 27 (21 days) patient had been given insulin on every day except 5 days. For the following reasons it seems certain that this insulin created hypoglycæmia every day that it was given:

(*a*) (1) Blood sugar on April 7 = 65 mgrm. per 100 c.c.
(2) ,, ,, ,, 9 = 45 ,, ,,
(3) ,, ,, ,, 19 = 35 ,, ,,

(*b*) Glucose (given by tube) caused her to awake from the comatose state, day after day, in about 20 minutes.

PART II.

On the twenty-second day events took a different course, as the following record shows:

Twenty-second Day. April 27, 1937.

(1) 8.30 a.m.: 40 units insulin.
(2) 1.30 p.m.: Glucose per nasal tube.
(3) *Clinical notes.*—11.20 a.m.: Very loud stertor; perspiring profusely; rapid pulse.

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11.27 a.m.: Still some stertor, which increased, so she was turned well on side, and hung over edge of bed; breathing improved.

11.35 a.m.: She was in quiet coma again. She remained in this quiet, comfortable coma until—

12.35 p.m., when she began to snore again. Held over bed and drained of saliva. This helped, but did not remove the stertorous breathing, which was still present at—

12.50 p.m., so she was again turned into bed and again went off into a quiet coma, with perfectly satisfactory pulse and respiration.

1.10 p.m.: Stertor, fairly rapid pulse and general trembling of muscles began.

1.20 p.m.: A similar attack with shivering (mild rigor) set in, with, however, a pulse-rate of 110 per minute only. This eased off, but it was thought better to give glucose at—

1.30 p.m.

2.50 p.m.: She was not awake, but had stertorous breathing, and at—

2.55 p.m.: Cyanosis.

3.5 p.m.: Patient was given $\frac{1}{100}$ gr. adrenaline subcutaneously.

3.25 p.m.: 30 c.c. 50% solution of glucose was given intravenously.

3.30 p.m.: Nasal tube was passed, and it was noticed that the glucose given at 1.30 p.m. returned almost in full quantity. Reaction of fluid from stomach was neutral or slightly alkaline.

3.30–4 p.m.: Another nasal feed (4 oz. glucose) was given; more intravenous glucose was administered—in all 90 grm.

Neither this nor subsequent nasal feed of glucose (about 5 p.m.) revived the patient, who, most inexplicably, remained in coma.

5.30 p.m.: *Blood sugar—280 mgrm. per 100 c.c.*

Later a salt solution, per nasal tube, was given, to influence the pseudo-tetanic condition which had, by then, set in.

6.45 p.m.: Salt solution, together with glucose previously administered, was vomited.

7.45 p.m.: Respiration seemed to be failing, and artificial respiration was done for a few minutes, and $\frac{1}{60}$ strych. given hypodermically.

7.50 p.m.: In view of the high blood-sugar found at 5.30, *10 units of insulin* were tentatively given.

8 p.m.: Temperature (under arm)—102·2° F. She vomited again. Any relieving of the coma noticed after the insulin was temporary—for instance she coughed forcibly over the side of the bed, but soon lapsed into coma again.

9 p.m.: Temperature (under arm), 102° F.

8.30 p.m.: *Blood sugar—200 mgrm. per 100 c.c.*

9.30 p.m.: In view of above a further *10 units of insulin* were administered —without much effect on the coma, but—

10.50 p.m., *blood sugar* was down to *85 mgrm. per 100 c.c.*, but she was not very appreciably less comatose.

12, midnight: Anxiety, lest the drop of blood sugar to 85—1 hour and 20 minutes after the second dose of insulin—might continue progressively during the night and lead to dangerous hypoglycæmia, dictated the administration of 3 oz. of glucose per nasal tube.

This did not affect the prevailing coma very much, and she was reported as having slept from 2 to 5 a.m., and been "restless" thereafter. She was sufficiently conscious, however, in this restless period to drink half a pint of tea.

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April 28, 1937.

8 a.m.: Temperature (under arm)—98·4° F.

8.45 a.m.: Took ½ pint tea, in small mouthfuls.

9.15 a.m.: Asleep; breathing naturally.

10 a.m.: Restless; tossing about.

10.45 a.m.: Pupils small and fixed.

10.50 a.m.: 3 oz. glucose given per nasal tube; pupils dilated to half the normal size.

11.40 a.m.: Fed 1 pint milk, 2 eggs, and ostomalt (1 tablespoon).

12.20 p.m.: Coughing forcibly; tossing about and yawning.

12.25 p.m.: Pupils began to react to light (sluggishly) for the first time in 20 hours. Abdominal reflexes were absent; plantar reflexes were normal.

12.30 p.m.: Catheter specimen urine taken: Reaction, acid; sugar (Benedict's solution), ·1%; albumen, a trace; acetone, small amount; diacetic acid, none; sp. gr., 1·022; occasional pus-cells and hyaline casts.

1 p.m.: Sweating; yawning.

1.45 p.m.: Temperature (under arm), 98·6° F.

2 p.m.: Asleep.

2.45 p.m.: Lumbar puncture. C.S.F. under marked pressure, dropping to normal pressure when 30 c.c. had been removed; *sugar* (Folin and Wu's method), *360* (at least) mgrm. per 100 c.c.; Benedict's test—10 drops reduced 5 c.c. to a brick-red colour—that is, the sugar content was *over 2%*.

2.55 p.m.: Catheter specimen urine—sugar present—about ·25%.

3.30 p.m.: *Blood sugar, 154 mgrm. per 100 c.c.*

4.0 p.m.: Enema—stools looked normal in colour and consistency.

4.20 p.m.: Temperature (under arm), 99·2° F.

4.50 p.m.: 10 units insulin given intramuscularly; this did not seem to ameliorate the stuporous state; but there was no longer present the perspiration which had been noticeable when she was in the hypoglycæmic condition.

5.25 p.m.: Corneal reflex present, but sluggish; abdominal reflexes absent; plantar reflexes normal in type, but sluggish.

5.30 p.m.: Stomach washed out; old milk curds and egg remnants returned in washing; 1 pint normal saline left in stomach.

5.45 p.m.: Much more wakeful; restlessly throwing her arms about.

7.10 p.m.: 1 pint cold saline per nasal tube.

7.15 p.m.: Moved limbs around bed; yawned naturally; opened her eyes.

7.35 p.m.: Relapsed into drowsiness, breathing heavily, and perspiring; pulse-rate was 104 per minute—this seemed like a *hypoglycæmic reaction.*

7.50 p.m.: 6 oz. glucose given by nasal tube.

7.56 p.m.: Trembling all over; vomited a little of the glucose solution.

8.30 p.m.: In coma; no perspiration; quiet breathing; slow pulse-rate.

11.0 p.m.: Given 1 pint saline by nasal tube.

11.30 p.m.: Blood sugar, 190 mgrm. per 100 c.c.

11.45 p.m.: Catheter specimen urine showed a trace of sugar.

At this stage the impression grew that something must have gone wrong with her carbohydrate metabolism—that insulin could still reduce the blood sugar (this had been proved biochemically, the previous evening, by the reduction, as the result of 2 injections of 10 units each of insulin, of the blood sugar progressively from 280 (at 5.30 p.m.) to 200 (at 8 p.m.) and to 85 (at

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10.50 p.m.)'; but that the administration of glucose, to check the resultant hypoglycæmia from going too far, while doing so effectively, did not wake the patient, but caused persistent *hyper*glycæmia with what looked like pseudo-diabetic coma.

12.40 a.m.: 1 pint saline by nasal tube.

During the day the fundi were examined occasionally, and seemed normal.

The blood-urea was normal.

Kahn test on C.S.F. was normal.

April 29, 1937.

11.10 a.m.: *Blood sugar, 130 mgrm. per 100 c.c.*

12.10 p.m.: *C.S.F. sugar: 300 mgrm. per 100 c.c.*

No food all day except saline.

April 30, 1937.

12.50 p.m.: *Blood sugar, 154 mgrm. per 100 c.c.*

No food all day except saline.

May 1, 1937.

10.50 a.m.: *Blood sugar, 130 mgrm. per 100 c.c.*

May 2 and May 3, 1937.

No food except saline during these days.

May 4, 1937.

11.45 a.m.: *Blood sugar, 95 mgrm. per 100 c.c.*

12.10 p.m.: *C.S.F. sugar, 180 mgrm. per 100 c.c.*

Patient now began to make an uneventful recovery on a diabetic regime. During the next 10 days the carbohydrate content of her food was gradually increased.

Some highly interesting features of the induced disturbance of carbohydrate metabolism from which this patient suffered over a period of some days seem to merit discussion.

For instance at 11.10 a.m. on April 29 her blood sugar was 130 mgrm. per 100 c.c.; but the really striking feature was the extraordinarily high level of the cerebro-spinal fluid sugar, which, estimated an hour later, was no less than 300 mgrm. per 100 c.c. This persisting discrepancy between the blood and C.S.F. sugar content would appear to be unique, and we have failed to find any like instance recorded in the literature.

Ordinarily as blood passes through the choroid plexuses some sugar is passed over to the C.S.F. Apparently in this case so much sugar passed over or accumulated in the C.S.F. that at one time (April 28) her C.S.F. sugar at 2.45 p.m. was 360 mgrm. per 100 c.c., and at 3.30 p.m. on the same day the blood sugar was only 154 mgrm. per 100 c.c.

At 12.50 p.m. on April 30 the blood sugar was again 154 mgrm. per 100 c.c.,

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i.e., even higher than it had been 24 hours earlier, and this in spite of the fact that she meantime had had no food whatever. At 10.50 a.m. on the following day the blood sugar was 130 mgrm., and it was only 3 days later, i.e., at 11.45 a.m. on the morning of May 4, that her blood sugar had dropped to the normal level—it was then found to be 95 mgrm. per 100 c.c.

During all these days she was given no food whatever, her life being maintained entirely by normal saline solution administered by the nasal tube.

Yet, in spite of this starvation, the C.S.F. sugar level was still abnormally high on that morning (May 4), namely 180 mgrm. per 100 c.c.

SUMMARY.

This patient, for 21 days, underwent the ordinary insulin shock treatment for schizophrenia without untoward incident. Then rather suddenly, on the twenty-second day, she did not awake from the hypoglycæmic comatose state on the administration of glucose. On examination she was now found to be in a state resembling diabetic coma ; and it required persistent anti-diabetic treatment to bring her back to normal.

We are indebted to Dr. P. J. Irwin, Medical Superintendent, Limerick Mental Hospital, for permission to undertake this therapy and to publish the clinical notes on this case ; and to Miss Mai M. O'Brien for her help with the technical side of the investigations.

Reference.—(1) Salm, *Münch. med. Wochenschr.*, Munich, July 2, 1937, lxxxiv, pp. 1046-9 (abstract in *Brit. Med. Journ.*, August 28, 1937).

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THE VALUE OF CONTINUOUS NARCOSIS IN THE TREATMENT OF MENTAL DISORDER.*

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" Nunc somno et inertibus horis
Ducere solicitæ jucunda oblivia vitæ."
—*Horace*, xi, s. 6, 61.

1. Introduction.

(*a*) HISTORY.

THERE does not appear in the literature any method of treatment to which the term " prolonged narcosis " can be applied prior to the year 1897, when Macleod (47, 48) published his first article on the bromide sleep treatment. The Hippocratic " Aphorisms " (126) mention that immoderate sleep is bad for the patient, but Celsus (129) quotes Asclepiades as being in favour of procuring sleep, and remarks that some insane patients recover if sleep is obtained, and Paulus Aegineta (128) observes that sleep produces oblivion of mental suffering and rectifies the distracted powers of reason. Aretaeus (127) in discussing satyriasis mentions " very protracted sleep " as the proper cure for the condition ".

Amongst the Arabians Avicenna (131) associates too much sleep with " grave maladies like apoplexy, paralysis and nightmare ", and therefore advises against allowing it to occur. Boerhaave (130) advised that sleep be procured in the treatment of melancholia, and Griesinger (132) advocated prolonged opium therapy, although stating that he aims at " an increasing quietening of the patients " and not " profuse sleep ". He also mentions the temporary remission of melancholia and mania following the narcotic effect of chloroform.

In a survey of contemporary means for inducing sleep in the treatment of insanity, Williams (133) (1845) quotes Andral and Van Swieten as having seen maniacal states cured by accidentally large doses of opium. Sir T. Clouston (134) mentions keeping " numbers of cases under (the influence of chloral) day and night for weeks, and many of them certainly got well " ; he then adds, " but I do not believe so much in it now ".

In 1897 and 1900 Macleod (47) first published the successful cure of cases

* A thesis approved for the degree of M.D. in the University of Manchester.

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of opium addiction and cases of acute mania by administering large doses of bromide, with the result that the patients passed into a condition of prolonged narcosis for periods of a week or longer.

In 1901 O. Wolff* published the results of treatment of confusional cases by trional, and in 1915 Epifanio* utilized luminal, but no general interest was taken in efforts to induce prolonged narcosis until Kläsi (7, 30), in 1922, first published his work on schizophrenics at the Burghölzli in Zurich.

Since that date interest has gradually increased. Müller (2) in 1925 gave an exhaustive survey of the literature up to that date. Lutz (46), in 1927, Cloetta (60) and Maier (61), in 1934, and finally Monnier (69), in 1936, have published a series of articles giving the experiences of the Zurich workers.

The treatment found its earliest supporters in Holland (1, 42), but in this country the first reference is in an article in the *Lancet*, 1926, by Dawson and Barkas (12), who reported unfavourably on it. Meerloo, of Santpoort, published a monograph on the subject in 1932, the substance of which appeared in the *Journal of Mental Science* in April, 1933 (1). Later came a series of contributions from the Cardiff workers (3, 4, 5, 24, 44), in which the use of insulin was advocated as a method of combating the toxic complications.

(*b*) The Present Conception of the Term "Continuous Narcosis".

The terms "continuous narcosis" and "continuous sleep" are usually employed to describe the method introduced by Kläsi in 1920 (7), in which the aim is to procure as much sleep as is possible by means of drugs, over a period of at least five days. The amount of sleep varies considerably, and it is only amongst the more recent publications (46) of the Burghölzli, in which sleep-charts are included, that it is possible to gather precise information. Periods of sixteen hours out of the twenty-four are considered adequate by some authorities (16), whereas the publications referred to above (46) describe periods of twenty-two hours. The term "continuous sleep" is in any case strictly speaking inaccurate, as the authorities allow some period for the nursing attention of the patient. An exception to this statement is the work of Macleod (47), who produced continuous periods of sleep amounting to five days and longer.

It is difficult therefore to distinguish between simple efforts by means of routine sedatives and the modern techniques, unless one adopts an arbitrary figure, such as sixteen hours, or unless one takes into consideration all the details of the case management, such as the approach to the patient, his isolation in a quiet room, and the subsequent handling of the case.

It is, of course, quite misleading to speak of the "somnifaine treatment",

* Quoted by Palmer and Paine (58*a*), Ström-Olsen (5), Lutz (46). O. Wolff, "Trional Kur", *Zentralbl. f. Neur. u. Psych.*, 1901, xxiv, p. 281. G. Epifanio, "D'ipnosi farmacologica prolongata applicazioni pro la cura di alcuni psychopathica", *Riv. di. Pat. Nerv. e. Ment.*, 1915, xx, p. 233.

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since this drug is by no means essential to the treatment and has been discarded by many authorities, including the Burghölzli.

(c) The Therapeutic Aim.

The treatment was introduced by Kläsi (7, 30) as a result of the teaching of Prof. Cloetta on the subject of local healing of wounds. Cloetta suggested that in the case of an ulcer, centripetal impulses passed to the spinal cord, and reflexly set up centrifugal impulses which impeded the procees of healing, but suggested that local anæsthesia would break this vicious circle.

Kläsi thought that there might possibly exist a similar vicious circle in the case of mental disease, in which affective excitation caused motor restlessness, which in turn caused further motor excitation. By employing central anæsthesia Kläsi thought that he could break this vicious circle.

This hypothesis of Kläsi is attractive, especially in certain agitated conditions, but unless given a wider meaning will not explain all the apparent benefit in some cases, and would, of course, exclude cases in which motor restlessness was not occurring.

Although first introduced by Kläsi to meet the demands of a hypothesis the treatment is largely used quite empirically, and for a much wider clinical field than he had envisaged; and we find Meerloo (42) saying in 1930: "We do not know beforehand which cases are likely to benefit most".

It is perhaps important to realize that the narcotic drugs used produce pharmacological effects other than those of sleep, and to consider the possibility that it is these other effects which are curing the patient. On the other hand, it may be the sleep which is the beneficial agent, or possibly a combination of sleep with other pharmacological effects.

The following would appear to be the possibilities which require consideration:

1. The sleep produced.
2. The narcotic effect on the cortex.
3. The narcotic effect on the subcortical region.
4. The narcotic effect on the co.tical-subcortical balance.
5. The effect of sudden withdrawal of narcotics after their prolonged use.
6. A specific barbiturate effect.
7. The facilitation of psychotherapy produced by the sleep.

These points will be dealt with later on under the general discussion of results, but they have been mentioned here in order to serve as an introduction to the next section on the choice of drug employed. Meanwhile it is probably correct to say that the aim of the treatment, as understood by the clinician, is to produce as many hours of sleep as are consistent with the safety of the patient, over a period of between ten days and a fortnight.

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(*d*) The Methods Employed.

Kläsi (7) originally utilized somnifaine, a mixture of the diethylamine salts of diethyl and allyl isopropyl barbituric acids in an alcohol-glycerol-water solution, and since that date the majority of investigators have utilized one or other of the barbiturates.

Mention has already been made of bromides (Macleod) and trional (Wolff). Wiethold (72, 73, 62, 74) utilized paraldehyde and Meerloo has evolved a "solutio barbamidoni".

In recent work (60, 61, 69) at Zurich "cloettal" has been used, which has the following ingredients:

Paraldehyde	0·4864	grm.
Amylene hydrate	0·1593	,,
Chloral hydrate	0·1157	,,
Alcohol (92%)	0·1747	,,
Allyl isopropyl barbituric acid	0·0409	,,
Digalen	0·0330	mgrm.
Ephedrine hydrochloride	2·4600	,,

per c.c.

This mixture represents a departure from the use of barbiturates alone, and has been created partly in accordance with E. P. Pick's (117) classification of hypnotics into two groups, viz., a group acting primarily on the cortex and one acting mainly on the brain-stem.

This grouping is not accepted by Sollmann (105), who criticizes Pick's conclusions as being based on ambiguous results and therefore not sufficiently justified. Sollmann also criticizes the report of Keeser* (1927), who found a greater concentration of the barbiturates in the mesencephalon than in the brain generally. Sollmann suggests that even if these experiments were to be confirmed, they would merely be an expression of the ratio of white and grey matter in the mesencephalon and cortex, rather than an expression of the selective action of the barbiturates on the mesencephalon.

However, in the published reports of post mortems on cases dying from barbiturate poisoning, it is frequently found that reference (1, 21, 95) is made to small lesions in the mesencephalon and pons, and these findings might appear to support the statement that the barbiturates had a selective action on the brain-stem.

This point is obviously one of considerable importance, and has to be considered in conjunction with the hypotheses concerning the focal origin of mental disorder. Situated in the mid-brain (121, 122, 123, 124) are important vegetative nervous centres, and if disorder in this region is a factor in mental

* *Arch. Exp. Path. Pharm.*, 1927, cxxv, p. 251.

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disorder, it is clear that any drug having its point of action on the mid-brain will be the drug of choice.

It would be of considerable value to treat two large series of patients by means of continuous narcosis, utilizing paraldehyde (a member of Pick's cortical group) for one series, and a barbiturate (a member of Pick's subcortical group) for the other, and to compare results. If the two groups were large enough, and the other experimental factors controlled as carefully as possible, it would be interesting to see which series produced the greater number of clinical recoveries.

In this series of cases it was decided to follow the principle underlying the choice of constituents of "cloettal", i.e., to combine a "cortical" with a "sub-cortical" sedative. In this way it is certainly more easy to produce satisfactorily the sleep required and it appears to be much safer, in that a smaller amount of barbiturate is employed.

If in addition, as seems likely, both cortical and subcortical factors are involved in mental disease, and if there proves to be any foundation for the classification of Pick, then such a technique whereby members of both groups are employed, will clearly be preferable.

This subject will be discussed more fully later on, under "Discussion of Results".

(*e*) The Role of Insulin in Diminishing Toxic Complications.

There have been various theories put forward concerning the mode of action of narcotic drugs, which have been summarized by V. E. Henderson (112, 116), and as these arose in part from physiological experiments concerning the metabolism of nervous tissue, it is perhaps permissible to refer very briefly to some of them.

In 1847 Bibra and Harleus suggested a relationship between the fat solvency of narcotic substances and their narcotic power, and in 1875 Claude Bernard propounded his precipitation or reversible coagulation theory.

Dubois (1882) sought to explain narcosis on the basis of a dehydration effect, and Richet (1893) correlated narcotic effect with water solubility. Winterstein (1900) thought that narcosis was essentially associated with asphyxia, and Traube discussed the possibility of an alteration of surface tension as the basis of the phenomenon.

The Meyer-Overton theory is, however, the one which has attracted most attention. In this theory attention is drawn to the solubility in lipoids of narcotic substances, and it is held that all chemically indifferent bodies which are soluble in lipoids must act as narcotics to living protoplasm, in so far as they can become distributed within it. If follows that the action of narcotics must appear first and most markedly in the cells in which lipoid substances predominate, and their relative activity must in turn depend on their mechanical affinity to lipoids on the one hand, and water on the other,

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and consequently on the partition coefficient which determines their distribution in the mixture of water and lipoids. In discussing this theory Sir W. Willcox (102) suggests that the hypnotic action may depend either on the prevention of afferent stimuli reaching the cells, or on a simple reduction of vital activity of the nerve-cells. This latter point has received considerable attention, especially the ability of brain-tissue to oxidize carbohydrates, and in a series of researches undertaken at Cardiff, notably by Quastel and Wheatley (24, 100, 114, 118), it has been shown that narcotics specifically inhibit the oxidation by the brain of substances important in carbohydrate metabolism, e.g., glucose, lactic acid and pyruvic acid. Moreover, the same workers have demonstrated a parallelism between narcotic power of a drug and its inhibitory effect on carbohydrate oxidation of brain-tissue, and they suggest, therefore, that narcosis depends on an interference of carbohydrate metabolism.

Ström-Olsen (3, 4, 5), also working at Cardiff, was impressed by the frequency with which ketone bodies appeared in the urine when toxic complications occurred in patients undergoing prolonged narcosis, and correlating these two sets of observations introduced the use of insulin-glucose to combat these complications.

Among 14 patients receiving the insulin and glucose only 1 case developed ketonuria, which disappeared on increasing the insulin to 30 units daily, and no cases required cessation of the treatment on account of complications. In contrast, ketonuria developed in 6 of the control series and treatment had to be suspended in 5 of these, and in only 4 cases was it possible to complete the course of treatment.

This work (4) was first published in 1933, and since then there has been very little work published which either confirms or rejects these findings, with the exception of that of Parfitt (22), whose series of carefully-treated patients showed alarming toxic symptoms, which occurred as commonly in those who received insulin as in those who had only glucose in addition to the narcotic somnifaine.

In this research it was decided to give insulin and glucose as a routine, closely following the instructions of Ström-Olsen, in order to ascertain whether this resulted in the freedom from toxic complications which that author has suggested.

Meanwhile it is interesting to recall a parallel series of observations concerning the value of insulin-glucose treatment in diphtheria. Schwentker (119), Noel and Benn (115) all came to the conclusion that insulin-glucose treatment lowered the mortality in diphtheria patients, whereas Begg (113), in 1935, denied this, and moreover pointed out that the alterations in the blood-sugar curve which occurred were not influenced by the giving of insulin. He suggests that though the diphtheria toxin evidently affects carbohydrate metabolism, this alteration is not influenced by insulin.

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2. Survey of Present Position Regarding the Treatment.

The treatment received its first survey by Müller (2) (1925), who drew attention to the high mortality-rate of 5%. He summarized his findings by stating that in his opinion the treatment had not come up to expectations, but that it " achieves results which cannot be obtained in any other way ", though " these results are only obtained in cases with a favourable prognosis ".

The experience of the Burghölzli Hospital is published in a series of articles by Oberholzer (10) (1927), Lutz (46) (1929), Cloetta (60) and Maier (61) (1934) and Monnier (69) (1936). These articles reveal the search for the ideal narcotic or narcotic mixture, and the refinement of technique whereby the patient is given the full benefit of complete isolation and careful nursing by nurses trained in psychiatric principles. The initial exclusive use of a barbiturate is finally supplanted by the mist. cloettal which, embodying the conceptions of Pick (117) and Molitor regarding cortically acting and subcortical sedatives, achieves a balance in which its sponsors claim an equal effect from the barbiturate and non-barbiturate components.

In Holland Meerloo (1) evolved a mist. barbamidoni which contains luminal and dial, together with pyramidon, atophan, caffeine and strophanthin. In the U.S.A. Palmer and Paine (58*a*) used sodium amytal. This drug originally attracted considerable attention in connection with a quite different technique. This technique (49, 51, 52, 54, 55, 56, 57, 58, 62, 66, 77, 78) employs the lucid interval which frequently follows the administration of the drug in patients suffering from such conditions as catatonic stupor, to gain psychiatric information regarding the patient's mental life. By this means a method is provided whereby the patient can be approached.

In this country the only published account of any considerable series of cases in which a drug other than somnifaine has been employed is that by J. S. Horsley (82), who utilized soneryl.

Although the treatment has received a fair degree of attention in the journals, the majority of text-books make little mention of it (27, 135, 136, 137, 138, 140, 142, 143). Thus, in the latest edition of Henderson and Gillespie's text-book (142) this opinion is expressed: " The advantages of somnifaine and similar methods in the psychosis appear to be more administrative than therapeutic ", and further, " We have seen unfortunate results. The Cardiff findings await confirmation ". On the other hand, M. Bleuler (13), in a review of the work of his father, Prof. E. Bleuler, in 1931, commends continuous narcosis as " an important aspect of therapy ".

As might be expected from his previous publications, M. Muller, in 1936, in his book on *Prognosis and Therapy in Mental Disorders* (138) states: " Continuous narcosis constitutes an indispensable therapeutic method for the psychiatrist ", and also that the treatment " stands to some extent (*einigermassen*) as an assured (*gesichert*) method ". Devine includes a

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favourable description of the method in the latest edition of his *Recent Advances in Psychiatry* (141).

J. Lange, however, in his 1936 *Concise Textbook of Psychiatry* (135), a standard recent exposition of orthodox German psychiatry, dismisses the method in a few words by saying: " It must be reserved for those physicians who think they cannot manage without it ".

In February, 1937, for the purposes of this research, a letter was addressed privately to the two leading American, German and French psychiatric clinics. Both American clinics replied saying that continuous narcosis was not in use at either hospital, and that, moreover, there was no hospital in the United States in which it had established itself. Similarly one German clinic replied that they had found the treatment of no use in the neuroses.

The actual results claimed by different authorities are extremely difficult to compare with one another owing to the differences in diagnostic terms used. There is a fair measure of agreement that the ideal period of treatment should be from ten to fourteen days, and a general similarity in the techniques employed, but the Zurich school are almost alone in advocating rectal administration of the sedatives, and the Cardiff school have been the pioneers in advocating insulin and glucose administration.

In summing up his attitude towards the problems involved in 1933 Meerloo (1) stated that:

1. The best narcotic agent was not determined.
2. The most favourable field and the most favourable phase are both undetermined.
3. Success can never be foretold.
4. Contra-indications are not adequately known, but that " there is no doubt that it produces results ".

3. Scope of this Investigation.

The purpose of this research was to produce experimental clinical data which could be used as a basis for testing the claims made on behalf of the treatment.

No patient was given the treatment in whom there appeared to exist an immediately favourable prognosis, and the case material was selected from amongst the patients admitted to a county mental hospital and a hospital for functional nervous disorders.

A detailed clinical record, including sleep-charts, was kept of each case, and it was hoped that an analysis of the data thus obtained would furnish a contribution to current efforts which are being made to achieve a safe and effective technique, and to determine the type of patient and the form of illness for which the treatment is most valuable.

In particular each patient was given insulin and glucose with every dose of

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somnifaine as a routine, according to the instructions of the Cardiff workers, and it was hoped that the study of the complications met with, together with a study of the manner of their appearance, would yield evidence which would be useful in assessing the protective value of the insulin-glucose methods.

Finally it was hoped to contribute to the existing theories concerning the mode of action of the treatment.

4. Technique and Case Management.

(1) Case Selection.

It is unusual to subject patients under the age of 16 or over the age of 55 years to the treatment, and in this series of cases only two such cases were treated.* Cases showing signs of organic brain disease were excluded, and, in general, all patients were considered to be in fairly good general health and condition. Cases showing marked arteriosclerosis were excluded, not only on account of the probable impairment of myocardial efficiency which its presence would imply, but also because these cases have a relatively poor prognosis. Some authorities exclude all cases showing any arteriosclerosis whatsoever, but in this research, provided the patient was in otherwise good condition, cases showing a slight degree of this complication were sometimes included. Particular attention was paid to cardio-vascular efficiency, and no case showing valvular disease of the heart, or in whom the heart was dilated, was given the treatment. No patient was given the treatment who showed any evidence of renal disease, and no patient in whom the presence of adventitious sounds in the chest were detected was given treatment in the first instance, or proceeded with if this complication developed.

Marasmus is mentioned by other authors (1, 10, 46) and is of particular importance, as it frequently happens that a patient is admitted to hospital who appears to be in fairly good physical condition, but who has not been taking sufficient nourishment.

Some authorities (Ström-Olsen (5)) do not consider well-compensated valvular disease of the heart a contra-indication, but this author lays great stress on excluding cases in whom there appears to be any evidence of myocardial degeneration.

It is inadvisable to give the treatment during the menstrual epoch.

More detailed observations regarding the choice of cases from the point of view of their mental condition will be found in Section 5. No case was submitted to the treatment for the purpose of this research in whom there appeared a probability of early recovery; whereas, on the contrary, one or two patients were given the treatment in whom prognostic indications were poor. The aim was to obtain results which would test the validity of the claims made for the treatment.

* Both these cases (R. 5 and R. 13) developed complications necessitating the cessation of treatment.

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The permission of relatives was always obtained in addition to that of the patient, unless he was too ill to have the treatment discussed with him.

(2) Duration of Treatment.

The majority of the patients were given a ten days' course of treatment. This is the period advised by the Zurich workers in their latest publications (69). Kläsi (7) originally gave an average of seven days, Muller (2) seven days, Palmer and Paine (58*a*) nine days, Segerath (8) twelve days, Ström-Olsen (3, 4, 5) fourteen days, Meerloo (1) fourteen days. Broder (77) (1936) waits until a "best day" in the course and disapproves of a standard period, maintaining that this more flexible method enables the physician to choose the best time for psychotherapy. Monnier (69), on the other hand, is of opinion that there is a distinct somatic factor at work, and he is one of the few authors to suggest a differentiation of duration of treatment for the different disorders, inclining to the belief that cases of schizophrenic illness should be given a longer course of treatment than cases of manic-depressive illness.

(3) Choice of Room.

The room must be rendered as quiet as possible and insulated from noise, but where the services of a special nurse are not available it must also be within convenient reach of the ward sister. By effective insulation of the patient from the routine noises incidental to a normal hospital life, it is possible to reduce the amount of sedative required and thereby render the treatment very much safer.

(4) Choice of Staff.

A special nurse should be assigned to the patient, and the personality of this nurse is of great importance in the majority of the cases treated. The Zurich workers (69) are amongst the few authorities to mention the allocation of special nurses during the post-narcotic phase of the treatment, but none of the authors appear to have considered the advisability of considering the personality of the patient in relation to that of the nurse during the treatment. An air of cheerfulness and capability probably creates the best atmosphere, and this is particularly true in the latter stages of the treatment, at which time the patient is most responsive to those around him.

(5) Reception of the Patient.

Before commencing the treatment good *rapport* is established with the patient, unless his condition renders this impossible, and this is most easily achieved by a careful case-history and a thorough physical examination. It is inadvisable to discuss the treatment with him more than twenty-four hours in

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advance, as it frequently occurs that he will become worried and apprehensive about it and get erroneous notions concerning what is going to happen to him. The night before treatment commences he is given a high enema.

He is placed in the treatment-room the same day on which treatment is to be commenced, and is not allowed to see any visitors or to receive any letters for fourteen days. In many cases visitors are kept away from the patient for a further fortnight, making a month in all.

During the treatment his room is kept darkened by curtains, except for two hours daily, during which nursing attention is being given and the room is aired. These measures effectively reduce the amount of narcotics required.

(6) Administration of Drugs.

The somnifaine, insulin and glucose are ordered under the medical officer's signature, and the sister or nurse in charge is given complete discretion to decrease, delete or delay the amount of somnifaine. If she feels that the time of the dose should be brought forward she must obtain the medical officer's permission before doing so. As soon as the patient awakes the medical officer is informed, and a dose of paraldehyde of from one to two drachms at a time is administered. In addition to this, paraldehyde is frequently ordered in combination with the somnifaine. This technique usually means that the patient will sleep from 10 o'clock in the morning until between 2 and 3 in the afternoon, when he will be given a dose of paraldehyde, and will sleep until about 5 o'clock. The nurse in charge is given complete responsibility with regard to restoratives, and informed that she must not hesitate to waken the patient to ascertain the depth of sleep which exists, or to administer coramine or cardiazol if she feels uneasy about his condition.

No patient is allowed to show a blood-pressure systolic reading below 90 mm. Hg., without restorative being given immediately.

In some cases it is found impracticable to give paraldehyde by mouth and then it may be given by the rectum, and if this is impracticable the drug may be injected deep into the muscles. Somnifaine must be injected deep into the muscles, as if it is given superficially it may cause a small abscess to develop which takes a long time to heal.

A strict aseptic and antiseptic technique is observed.

All patients receive as a routine their morning dose of somnifaine at 9 a.m. and their evening dose between 4 p.m. and 7 p.m. This routine enables a daily estimate to be made of the amount of sleep that is being procured, and it seems probable in consequence that this is a much safer method than that advised by many of the authors, which is to give the somnifaine whenever the patient awakes. In practice the wakefulness of the patient is a most unreliable indication of the state of his intoxication with the barbiturate, and it is probably wisest never to inject a dose of somnifaine at a period of less than eight hours

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from the previous dose. If the patient is wakeful in the intervening period he should be kept asleep by means of paraldehyde. This rhythm of treatment guards against the likelihood of cumulation. Moreover, although the Zurich workers advise against this routine, a careful examination of their published charts (46) makes it appear that in practice the actual times at which they administer their drugs correspond fairly closely with the times advocated in this research. However, in considering these two modes of administration of the narcotic drugs, it is important to point out that whereas in Zurich and with the majority of other authors, a single drug, or as in the case of "cloettal" a standard mixture of drugs has been employed, in this research it has been my experience that a much safer and more effective technique is to employ paraldehyde and the barbiturate separately. This technique aims at a more or less fixed quantity of barbiturate and a variable amount of paraldehyde, and since the paraldehyde is given as occasion arises, it might be considered that as regards the procurement of sleep the two techniques are essentially the same. What does constitute an important difference, however, is that in the technique employed in this research far greater safety is obtained on account of ensuring an adequate interval between one dose of barbiturate and the next. Nevertheless it will occasionally be found helpful to inject a third dose of somnifaine about 1 o'clock in the morning.

(7) Nursing.

The technique outlined above allows two main periods for nursing attention, namely, between 8 and 9 in the morning and 5 and 6 in the afternoon. During these periods the nurse takes the patient's temperature, pulse, respiratory rate and blood-pressure, and examines the urine. These findings are all charted, together with the amount of sleep obtained. Attention is then given to the patient's bowels and bladder, and he is given nourishment and fluids. He is got out of bed and assisted to walk at least twenty-five paces along the corridor, and during this period engaged in therapeutic conversation. The aim of the nurse is to make simple suggestions that he is improving, and towards the end of the treatment she endeavours to introduce a cheerful state of mind in the patient during his wakeful periods. Each patient is given a minimum of 60 oz. of fluid in the twenty-four hours, and, if possible, as much as 100 oz. is given. The fluids consist of milk and sweetened lemonade or barley-water, and in the milk the patient receives two eggs daily, together with ¼ oz. of butter. When necessary an aperient is added to these fluids, and no patient is allowed to go more than twenty-four hours without moving his bowels or receiving an enema. The urine is tested twice daily for albumin and ketone bodies, and the total quantity passed in twenty-four hours is measured. A careful watch is kept on the bladder, but catheterization is avoided as far as possible, as these patients are very liable to urinary infection.

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The patient is nursed in a pneumonia jacket, and carefully protected from draughts and chills, and no nurse is allowed to go near the room who has a cold, nor is any treatment given if there is any ward infection, such as influenza. The patient's chest is given friction twice daily with a tepid sponge, and in older cases the dorsal region is rubbed with camphorated oil. An ordinary high bed is employed for the majority of cases, though occasionally, especially in those cases who develop a psychotic phase, it is necessary to nurse them on two mattresses on the floor. A cylinder of oxygen is kept in the patient's room, and a small hypodermic syringe, together with a box containing ampoules of coramine, is always available. The patient is never allowed to lie for more than six hours in one position, and is encouraged to go to sleep sometimes on one side, sometimes on the other, sometimes on his back, and particularly quiet patients may be given inhalations of oxygen 95%, carbon dioxide 5% for three minutes twice daily. The patient must be seen at least once every quarter of an hour, and if he is causing any anxiety a special nurse is allocated to watch him.

The general appearance of the patient, the quality of the respirations, and the blood-pressure, are considered to be the most valuable guides to his condition.

The patient is nursed in a room in which the temperature is kept at approximately 65° F. by central heating.

(8) Complications.

The various authors have described approximately fifty different complications, but in this series the majority of these were never met with, and it is clear from reading in the literature that considerable variations in importance are to be attached to those which are mentioned. Some of the complications described would appear to be not so much true complications, as rather natural accompaniments of the prolonged use of narcotics. To some extent, however, they are valuable in helping to assess the degree of intoxication which exists at any one moment. Such, for example, are the various signs of motor inco-ordination and the diminution of physiological reflexes, especially the cough and swallow reflexes. Others of these complications would appear to be an expression of tissue reaction to the barbiturates. These are, on the whole, uncommon, and do not, as a rule, indicate impending collapse of the patient, but they generally require the treatment to be proceeded with cautiously. Retention of urine, bradycardia, extreme flaccidity of the muscles and transitory œdema, are examples in this class.

In a third group must be mentioned a set of complications which are really to be regarded as accidents, and are not directly due to the treatment itself. Such, for instance, are peripheral nerve palsies and fractures.

Finally there are the true complications, which mean either that the patient is intolerant of barbiturates or that he has been given doses in excess

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of his tolerance. The complications in this group are the most important, and a careful watch must be kept by the nurse in charge for their occurrence. When they occur they usually require the administration of antidotes, and indicate that the treatment must be proceeded with more slowly. Their persistence usually necessitates the termination of the treatment. This group includes shallow, laboured breathing, especially where the respiratory-rate is greater than 25 per minute ; pyrexia, especially temperatures greater than 99·6° F. ; nausea and vomiting, especially when the patient vomits more than twice in one day ; a fall in the systolic blood-pressure below 100 mm. Hg., and especially should it fall below 90 mm. Hg. ; tachycardia greater than 90 beats per minute, and especially where it exceeds 100 beats per minute ; any tendency to cyanosis ; oliguria amounting to less than 30% of the total fluid intake ; albuminuria and ketonuria, except very slight traces ; cardio-vascular impairment, which may show itself first by a pulmonary hypostasis ; finally, a very rare complication mentioned by Meerloo, jaundice.

List of Complications Mentioned by Various Authors.

Common complications.	Toxic complications.	Complications involving particular tissues.	Accidents.
Inco-ordination ; nystagmus ; hypo-ventilation of lungs ; diminution of swallow and cough reflexes ; dysphagia ; coma ; nausea with occasional vomiting.	Shallow laboured respirations ; extreme restlessness ; extreme drowsiness with small doses ; pyrexia ; hypopyrexia ; persistent vomiting ; sudden death.	Primitive reactions ; psychotic phase ; epileptiform convulsions ; delirium ; retention of urine ; bradycardia ; extreme flaccidity of muscles.	Peripheral nerve palsies ; fractures ; respiratory inflections ; inhalation ; pneumonia.
Fall in blood-pressure down to approximately 100 mm. Hg ; tachycardia up to 90 beats per min.	Fall in blood-pressure below 90 mm. Hg. ; tachycardia more than 100 beats per min. ; cyanosis ; collapse ; pulmonary hypostases.	Transitory œdema.	
Oliguria.	Anuria ; albuminuria.		
	Ketonuria ; exanthemata.	Dermatographia.	
	Jaundice.	Meteorism.	

All the above complications have as their ultimate complication cardio-vascular collapse, and this is usually ushered in by a pronounced fall in the blood-pressure, a rapid thready pulse, pallor, rapid laboured respirations, and, if the patient is awake, a sense of impending catastrophe with chilliness.

Special mention must be made of pneumonia, which most authorities single out as the most important complication. Broncho-pneumonia may occur

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from inhalation of food and lobar pneumonia may occur as a result of hypostatic congestion. When this occurs there are four factors which require consideration. Firstly, the depression of the respiratory centre in the medulla; secondly, the impairment of the pulmonary circulation; thirdly, the possible pericapillary toxic effect of the barbiturates rendering the lung parenchyma more susceptible to infection; and finally, the effect of persistent lying in one position.

In this series the most valuable measures of the patient's general condition were found to be the quality of the respirations and the blood-pressure. A detailed analysis of the complications encountered is given in Section 5.

The treatment for all the above complications is to administer more fluid, to postpone the next dose of somnifaine and reduce the dose to be given, and where there is any threat of cardiovascular collapse to give immediately a dose of coramine or cardiazol. Where the extent of the complication has exceeded the limits mentioned above, the treatment should be stopped, at least temporarily, until the patient's condition has returned to a normal level, and in most cases the treatment can then be recommenced, though it may be necessary to reduce the amount of drug which has been given, and be content with a correspondingly less amount of sleep. It is never wise to proceed with the treatment of a patient over whose condition one feels worried. Monnier lays considerable stress on this attitude.

Cardiazol (pentamethylenetetrazol) and coramine (pyridine β-carboxydiethylamide) (107) appear to be of almost equal value, but the impression was formed in this research that coramine was perhaps the better of the two drugs. It is convenient to keep a stock solution of cardiazol—1½ gr. to ½ oz. water for oral adminstration—and to use the coramine subcutaneously. Coramine is a much more expensive drug, and this method is consequently preferable to using coramine alone in cases of minor complication. The cardiazol may be repeated until a total of 10 gr. has been administered within the hour, and the coramine may be given in cases of emergency in as high a dosage as 15 c.c. intravenously. Both these drugs act by stimulating the medulla and by producing peripheral vaso-constriction. It may be necessary to resort to more drastic methods, in which case lumbar puncture, and in the last resort cisternal puncture may be performed. The necessity for these methods must be extremely rare and will occur only in cases of extreme idiosyncrasy to the drug. In this series neither of these two latter procedures was ever found necessary, nor was it ever necessary to give more than 2 c.c. of coramine. In cases of circulatory collapse elevation of the end of the bed, hot-water bottles, and the administration of oxygen are all of service.

Monnier, Meerloo and other authors mention caffeine, strychnine, strophanthin, ephedrine, intravenous hypertonic glucose solution as other antidotes which may be used, and in "mist. cloettal" a routine dose of ephedrine and digalen is mixed with the narcotic drugs. Should it be decided with any

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particular patient to administer a routine vasomotor tonic, it is probably more useful to employ ephedrine than coramine or cardiazol, because although all three of these drugs have both a central action on the medulla and a peripheral action on the blood-vessels, the action of the ephedrine is more peripheral than central, and in consequence does not antagonize the narcotic effect of the treatment; whereas the action of the coramine and cardiazol is more central and does not act against the narcotic effect. On the contrary, when dealing with cases of threatened collapse, the central action of coramine and cardiazol is valuable. In this research vaso-constrictor drugs were not employed unless there appeared special indications, such, for instance, as a persistently low blood-pressure, and in the majority of cases the drug employed in this connection was cardiazol.

It will be seen that in the above account circulatory collapse is the danger most to be feared, and the Zurich workers attribute this in the main to peripheral vasomotor paralysis (69).

Reference has already been made in the Introduction to the work of Ström-Olsen and also that of Parfitt (22), and it is necessary to pay attention to the warning contained in a leader in the *Lancet* (No. 5873, p. 671, 1936) against "too easy confidence in such precautions", i.e., the routine administration of glucose and insulin. In this series insulin and glucose were given as a routine closely following the instructions of Ström-Olsen. The value of this routine will be given a full discussion later on (Section 6).

(9) Post-Narcotic Management.

In the first three days following the cessation of treatment the patient is intoxicated and should not be allowed out of his bed, but during this period intensive efforts should be made by means of suggestion and persuasion to facilitate his recovery. It is most important to discourage him from discussing his previous symptoms or his various worries, and towards the end of the week he may be allowed up gradually and be placed on a course of hydrotherapy and ultra-violet light. A course of aeration baths for fifteen minutes daily was found to be most valuable, and the patient should be put on a simple tonic mixture containing a little strychnine and iron. It is also most important to place the patient on a routine nocturnal sedative, as insomnia is so frequent a complaint of these patients that the value of the suggestion to them that their insomnia has been cured by the treatment is thus reinforced.

Ten days after the cessation of the treatment the patient, if he is well enough, is given therapeutic occupation for two hours daily, and in the afternoon it is extremely useful for the patient to be taken out for short walks with a suitable nurse who has been trained in psychotherapeutic principles, and where the physician in charge of the patient can manage to do this the results are usually excellent.

It is usually known whether the patient is going to improve as a result of

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the treatment by three weeks from the cessation of his period of sleep, and thereafter less intensive methods are pursued. It is important to instruct the patient's relatives or visitors to make short visits during the first month after the treatment, not more than once a week, and to ask their co-operation in not discussing any of the patient's difficulties with him, or referring to his previous symptoms. This practice is different from that advocated by the Zurich workers (46, 69) who, on the contrary, advocate the early visit of relatives, together with early discharge. I have seen unfortunate relapses which appeared to follow upon the visits of a patient's relatives, and one case of attempted suicide where a patient was mobilized and sent back to his work in accordance with the instructions of the Zurich workers. This divergence of opinion may arise because in this research a different type of case material was being handled, where the criterion of recovery was not discharge from hospital, but successful resumption of work.

In certain cases where it is proposed to employ more intensive psychotherapeutic methods it is usually advisable to postpone these until at least three weeks after the end of the treatment.

5. Statement and Analysis of Results.

The tables which follow set out the results obtained in this research. In choosing the descriptive label to be employed for the various headings it was decided to rely on four criteria.

By "recovery" is understood that the patient left hospital free from the symptoms for which he had presented himself for treatment, and in the majority of cases was considered fit to follow his employment, and did in fact return.

By "permanent improvement" is understood in the majority of cases that the patient's eventual recovery was due in considerable degree to the treatment. Patients R. 11 and R. 25 were suffering from a recurrent attack of mania. The treatment resulted in a considerable improvement, but neither of them were discharged from hospital. Patient W. 13 remained in hospital whilst arrangements were being made for his family's domicile in England.

By "temporary improvement" is understood that the patient showed improvement in his main symptoms following on the treatment and attributable thereto, but subsequently showed a complete relapse to his former condition. These cases of temporary improvement lasted from a few days to three weeks at the most.

By "no change" is understood that the treatment had no visible effect on the patient.

It will be seen that the criteria adopted here were somewhat strict, and it is important to consider in this connection the type of patient which was being handled. In the county mental hospital (which included all the "R" series), the patients were all of the rate-aided class, and, moreover, were drawn

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Table of Results.

	Not improved.				Permanent improvement.		Recovered.		Total treated.	Treatment broken off.	Total.
	No change.		Temporary improvement.								
Acute mania	..	..	..	..	R. 11	1	R. 21	1	2	..	..
Recurrent mania	R. 13	1	R. 7	1	R. 25	1	..	..	3	..	..
Depression	R. 3, R. 8, W. 24	3	R. 1, W. 2, W. 12	3	R. 22, W. 3 W. 10, W. 5	4	R. 18, R. 19, R. 23 W. 8, W. 16	5	15	..	..
Agitated depression	R. 12, R. 16, W. 18	3	W. 19	1	W. 13	1	W. 1, W. 17	2	7	..	..
Schizophrenia :											
Simple	..	..	..	..	R. 15	1	..	..	1	..	..
Hebephrenic	R. 14, R. 24	2	..	..	W. 4	1	..	..	3	..	..
Catatonic	R. 4, W. 20	2	R. 9	1	..	..	R. 10, W. 9	2	5	..	..
Paranoid	R. 17	1	R. 6	1	..	..	..	..	2	..	..
Anxiety states	..	..	R. 20	1	W. 6	1	W. 5, W. 7 W. 11, W. 14	4	6	..	..
Alcoholism	..	..	..	..	..	..	W. 25	1	1	..	..
Cases broken off within 5 days	R. 2, R. 5, W. 21 W. 22, W. 23	..	..	..	..	..	..	..	..	5	..
Total treated	..	12	..	8	..	10	..	15	45	..	..
Total broken off	..	..	..	..	..	..	..	..	..	5	..
Total	..	..	..	..	..	..	..	..	..	..	50

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from a district in which unemployment was rife, though it is not one of the "special" areas. Consequently if a patient did not immediately return to his work it did not necessarily imply an incomplete recovery. On the contrary, the patients in the "W" series were mostly of the middle class, and in many cases were owners of their own businesses, and were able to return to work under circumstances which permitted of their taking things quietly for a time. A particular case in point was Case W. 8.

Recovery-Rate.

It will be seen that a total recovery-rate of 33⅓% occurred for the 50 patients included in this series; this figure corresponds exactly with the figures given by Meerloo (1) for 500 cases. The recovery-rate was greater, as was to be expected, in the "W" series than in the "R" series, that is in the more recoverable forms of mental disorder. It is very difficult to make an adequate comparison with the results published by other authors, as the type of case handled varies in different countries. Moreover, considerable differences are bound to exist in the criteria which governed case-selection and classification of recoveries. This probably accounts for the difference in results published by the various authors. The figure given by Meerloo of 33⅓% out of 500 patients is probably the most valuable, as this author appears to have handled a fairly wide selection as well as a large number of cases. A chart is included showing the results published by fourteen authors (see p. 655).

Manic Depressive Group.	Palmer and Paine.	Parfitt.	MacNiven.	Ström-Olsen.	Hennelly.
Recoveries (Mania	5	2	1	11	32
Recoveries (Depression	1	3	3	6	
Considerably impressed	2	0	9	13	26
Some improvement	0	8	8	0	
No change	1	7	4	15	30
Total treated	9	20	25	45	88

Psychoneuroses.	Ström-Olsen.	Parfitt.
Recovered	8	0
Considerable improvement	0	2
Some improvement	2	2
No change	3	7
Total treated	13	11

Recovery-Rate of Sub-groups.

Manic-depressive Group.

Both cases of acute mania showed permanent improvement or recovery. Of the 3 cases of recurrent mania that were treated, 1 showed permanent improvement, whilst a second made a complete remission, but was probably

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Chart of Analysis of Recovered (R.) and Considerably Improved (C.I.), together with Full Totals below.

	Monnier.		Lutz.		Oberholzer.		Müller.	Kläsi.	Moser.	Demole.	Gjessing and Lingjaerde.	Palmer and Paine.		Ström-Olsen.		Parfitt.	Stuurmann.	Meerloo.	Kooy.
	R.	C.I.	R.	C.I.	R.	C.I.						R.	C.I.	R.	C.I.				
Schizophrenia :																			
Simple	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..
Hebephrenic	..	..	3	11	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Catatonic	4	3	3	13	3	3	..	..	..	..	..	1	1	0	3	..	..	..	..
Paranoid	..	..	..	..	0	2	..	..	..	..	..	..	..	..	..	..	..	..	..
Psychogenic	18	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Acute schizoid	3	0	..	..	1	2	..	..	..	..	..	..	..	0	8	..	..	..	..
Manic depressive forms	0	3	0	2	7	2	..	..	..	..	..	1	1	2	3	..	..	..	..
Autistic	..	..	..	..	..	..	..	..	..	..	..	..	..	2	5	..	..	..	..
Agitated	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Chronic	15	6	..	..	1	3	..	..	..	..	..	..	..	..	..	..	..	..	..
Undifferentiated	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Totals :																			
Recovered	40	..	6	..	12	..	..	6	..	..	..	3	..	4	..	6	..	33⅓% of all cases recovered.	..
Considerably improved	..	13	..	26	..	12	1	2	..	..	5	..	2	..	19	..	2		..
Some improvement	19		19		19		3	6	12	6	11	6		..		5	15		10
No change	39		15		35		15	9	11	17	10	2		26		8	21		14
Totals treated	111		66		78		19	23	23	23	26	13		49		19	38	500	24

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discharged from hospital prematurely and, as a result, relapsed again. The third case remained unchanged. Of the 15 simple depressive patients, 5 cases recovered and 4 made a permanent improvement. An analysis of the possible factors involved in the depressive group shows that habitus alone amongst the factors examined appears to bear a direct relationship to the question of recovery. In a comprehensive survey of the influence of various factors bearing on the prognosis of depressive illness A. J. Lewis* came to similar conclusions, with the important difference that he did not investigate the bearing of habitus. The agitated depression group appears to present as favourable a field as the group of simple depression, but the number handled (7) is too small to allow of any detailed analysis. When it is considered that this group contains on the average an older class of patient than the other groups the results appear most encouraging. Both the patients who recovered (W. 1, W. 17) would have required certification had the treatment not been available, and in one case (W. 1) the patient had recently been under "temporary" treatment.

Schizophrenia.

In this group only the catatonic variety produced recoveries. In the case of W. 9 the result was most unexpected and certainly prevented certification.

Anxiety States.

This group, as was to be expected, produced the highest recovery-rate, and in only one case was there anything less than permanent improvement. In this case (R. 20) the convalescence was not fully under the control of the physician and treatment had to be interrupted. The one case included as "permanently improved" actually recovered sufficiently to return to his employment. However, he suffered from a slight stammer, and in order to make certain of not overstating the result he is not included as a complete recovery.

Alcoholism.†

This patient (W. 25) is included as representative of a group of patients for whom the treatment proved itself invaluable by combating a state of restless indecision and unwillingness to admit the necessity for treatment. It rendered the patient more amenable, facilitated the establishment of a positive transference, and though the patient's eventual recovery was due to the psychotherapeutic methods which were subsequently employed, as well as to the successful manipulation of her economic problems, the sleep treatment undoubtedly enabled these measures to be successfully instituted.

* *Journ. Ment. Sci.*, 1936, lxxxii, p. 488,
† Cf. Forel, "Les Toxicomanies", *Ann. Med. Psych.*, 1931, lxxxix, 2, p. 362.

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Factors Bearing on Recovery.

In addition to the table showing the general recovery-rates of the treatment, tables are included to show the bearing which some other factors had upon the recovery of the patient.

Habitus.—Of particular interest is the one which deals with body habitus. It can be seen that approximately half of all the pyknic patients recovered; one-third of the athletics; and only one-fifth of the asthenics. If one accepts the findings of Kretschmer (139) regarding the association of habitus with schizophrenic and cyclothymic personality types, then these results merely reflect the conclusions of other authors (2, 3, 4, 5, 58*a*), that illnesses having a more predominantly manic-depressive colouring fare better under the treatment.

Table Showing the Bearing of Habitus on Recovery.

	Pyknic.	Athletic.	Asthenic.	Dysplastic.
No change	3	9	1	0
Temporary improvement	2	3	2	0
Permanent improvement	2	7	1	1
Recovered	6	8	1	0
Total	13	27	5	1
Percentage of total recoveries	46%	30%	20%	

Duration of present attack.—The chart shows a relationship between the duration of the attack and the prospects of recovery. However, the factors involved here are complex. An analysis of the depressive group shows no such relationship, and the findings in the general chart may be only an expression of the fact that the more recoverable forms of illness, such as the anxiety types, tend to have a shorter duration of illness than do the more irrecoverable types, such as the schizophrenic group.

Age.—These objections do not apply so completely to the age-factor, and the chart again shows a positive correlation. This shows a very small difference, however, which does not appear in an analysis of the depressive group. It is clear that a much larger series of cases require analysis to make any valid deductions.

Amount of sleep.—In this chart only slight differences appear, but seem to point to the tendency to longer periods of sleep in the recovered cases.

No attempt has been made to analyse the bearing of the family history and previous personality on the prospect of recovery, as it was felt that these factors were outside the scope of the present investigation, constituting as they do a large field of research in themselves.

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Average of 50 cases = 39 years.	Age. Average for all cases.	Median for all cases.
No change	41 years	40 years.
Temporary improvement	40 ,,	42 ,,
Permanent improvement	40 ,,	43 ,,
Recovered	37 ,,	35 ,,

	Duration of Present attack. Average for all cases.	Median for all cases.
No change	21 months	17 months.
Temporary improvement	16 ,,	15 ,,
Permanent improvement	12 ,,	6 ,,
Recovered	11 ,,	7 ,,

	Average number of hours sleep in 24 hours.
No change	16 hours.
Temporary improvement	16⅓ ,,
Permanent improvement	16¾ ,,
Recovered	17⅓ ,,

Complications.—A chart is included in which the majority of complications mentioned by the various authors are tabulated.* Many of these were not met with in this work. The complications encountered in this series, together with their frequency, are given, and another chart analyses the cause of the breaking-off of treatment. In 10 cases treatment was discontinued on account of the patient's condition. Of these 10 patients, R. 2, W. 21, W. 22, gave the impression of being intolerant to somnifaine. They exhibited persistent vomiting and became very ill, and in consequence objected very strongly to a continuation of treatment. Case R. 13 was a man aged 67, and treatment was suspended as a precautionary measure in view of the occurrence of pyrexia and tachycardia. Case R. 15 developed an unusual form of collapse. He suddenly complained of being cold and felt he was going to die. His pulse became rapid, but his blood-pressure did not fall. As this condition set in following on the giving of insulin a blood sugar estimation was performed, but there was no hypoglycæmia. Clinically the condition resembled an acute anxiety attack. Case R. 22 showed a tendency to low blood-pressure, and with the onset of pyrexia it was decided to suspend treatment, especially as he had already received seven days' treatment. Case R. 25 developed retention of urine which required catheterization. His treatment was suspended as a

* Pp. 660, 661.

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Depression Group.

	Case No.	Total hours of sleep.	Days.	Average of hours sleep per day.	Group average.	Previous attack.	Age.	Group average.	Duration of present attack (in months).	Group average.
No change	R. 3	116	10	11	15	..	43	40	1	5
	R. 8	175	10	17		1	55		9	
	W. 24	190	10	19		..	23		4	
Temporary improvement	R. 1	107	10	11	17	..	58	50	36	17
	W. 2	206	10	21		..	51		15	
	W. 12	204	10	20		..	42		1	
Permanent improvement	R. 22	124	7	17	18	..	47	42	6	4
	W. 3	181	10	18		1	50		4	
	W. 10	168	10	17		1	29		7	
	W. 15	190	10	19		..	43		1	
Recovered	R. 18	190	10	19	17	1	57	46	½	17
	R. 19	176	11	16		1	34		12	
	R. 23	187	11	17		..	51		36	
	W. 8	171	10	17		..	53		36	
	W. 16	65	4	16		..	35		½	

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precautionary measure. Cases R. 5, W. 9 and W. 22 were the only three that gave rise to any real anxiety. Case R. 5 was a man, aged 61, of asthenic habitus, who had a marked degree of arteriosclerosis; he gave no warning of his impending collapse, but responded rapidly to coramine and cardiazol. Case W. 9 collapsed in a similar manner, but there was no apparent cause for this. Case W. 22, however, was rather different. Following on her evening dose of somnifaine she passed into a deep comatose sleep, and the nurse became worried about her condition on account of her breathing, which was shallow and laboured. The medical officer was summoned, and found that the patient could not be roused. Coramine was administered, but it was some time before she roused herself. All these three patients were of asthenic habitus.

List of Complications Met with in this Research, together with their Frequency.

Complications.	Number of times encountered.
Tachycardia	12
Pyrexia	9
Persistent vomiting	7
Ketonuria	6
Low blood-pressure	3
Circulatory collapse	3
Bradycardia	2
Retention of urine	2
Hiccup	1
Coma	1
Occurred after treatment.	
Epileptiform seizures	1
Exanthemata	1

Cases Showing Minor Complications.

Reference.	Age.	Habitus.	Complications met with.
W. 2	51	Pyknic	Trace ketonuria.
W. 8	53	Athletic	Occasional hypopiesis.
W. 13	54	,,	Occasional poor colour.
W. 17	38	Pyknic	Became a little œdematous towards end of treatment beneath the eyes.
R. 10	18	Asthenic	Vomited and was pale on second day.
R. 16	55	Athletic	Pyrexia on fourth day; ketonuria on eighth day.
R. 19	34	Pyknic	Pyrexia on eleventh day.
R. 21	37	Athletic	Vomited occasionally.
R. 23	51	Pyknic	Tendency to hypopiesis throughout.
R. 24	24	Athletic	Scarlatiniform rash on eleventh day.

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Cases Showing Complications Necessitating Cessation of Treatment—10 Cases.

Reference.	Days of treatment.	Average daily dose of somnifaine.	Average daily hours of sleep.	Age.	Habitus.	Possible reason for complication.	Actual complication which caused cessation of treatment.	Day of occurrence.
R. 2	4	3 c.c.	10	37	Athletic	N.A.D.	Ketonuria, frequent vomiting and pyrexia	4
R. 5	4	4 ,,	14	61	Asthenic	Arteriosclerosis and poor nutrition	Circulatory collapse	5
R. 13	9	2·5 ,,	16	67	Athletic	Arteriosclerosis	Pyrexia with tachycardia	8
R. 15	5	3·5 ,,	17	25	Asthenic	N.A.D.	Ketonuria and collapse ; pyrexia	6
R. 22	7	2·5 ,,	17	47	Athletic	,,	Hypopiesis ; pyrexia	7
R. 25	8	3 ,,	12	48	Pyknic	,,	Retention of urine	6
W. 9	5	3 ,,	17	29	Athletic	? Poor nutrition	Circulatory collapse	4
W. 21	2	2 ,,	..	32	Athletic	N.A.D.	Persistent vomiting	2
W. 22	2	2 ,,	..	19	Asthenic	? Poor nutrition	Coma ; collapse	2
W. 23	3	3 ,,	..	41	Athletic	N.A.D.	Persistent vomiting	3
Averages	5	2·5 ,,	15	41				5th

8% of total : Pyknic patients (1 in 13).
22% ,, : Athletic patients (6 in 27).
60% ,, : Asthenic patients (3 in 5).

Cases that Caused Anxiety at Some Time of the Treatment, but with which the Treatment was Continued Successfully.

Reference.	Days of treatment.	Average daily dose of somnifaine.	Average daily amount of sleep.	Age.	Habitus.	Possible cause for complication.	Complications met with.
R. 4	11	4·5 c.c.	10½	26	Athletic	*Nil*	Cyanosis.
R. 7	11	4 ,,	12	47	Asthenic	,,	General health poor.
R. 8	10	3 ,,	17½	55	Athletic	,,	Hypopiesis ; cyanosis.
R. 14	10	4 ,,	17	21	,,	,,	Frequent vomiting ; ketonuria.
W. 4	10	4 ,,	17	21	Dysplastic	,,	Bradycardia throughout.
W. 6	9	3 ,,	16	31	Athletic	,,	Frequent vomiting with ketonuria.
W. 11	10	2·5 ,,	19	16	,,	,,	Frequent vomiting.

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Reviewing the relationship of habitus to these 10 patients, it will be seen that only 1 in 13 pyknic patients is included (8%) ; 6 in 27 athletic patients (22%), and 3 out of 5 asthenic patients (60%) developed toxic complications. Only the 3 asthenic patients gave rise to any real anxiety on account of their condition.

These serious complications tended to occur at the beginning of the treatment, especially between the second and fifth day. This is frequently mentioned by other authors, and probably reflects the relative inability of these patients to establish a tolerance to the barbiturates.

In a further 7 cases complications were met with which caused a certain degree of anxiety, but the treatment was continued successfully for the full period.

In a further 10 cases minor complications occurred which did not cause any real anxiety. Their presence was recorded only as a matter of routine.

In only 6 cases in the whole series of 50 were ketone bodies found in the urine, but these 6 included 2 in whom the treatment had to be suspended. However, in this series of cases insulin-glucose was given as a routine. It is possible, therefore, that ketonuria would have occurred with greater frequency had the treatment not been given. What is of interest, however, in reference to the main contention of the Cardiff school of workers is that of the 10 cases in whom treatment had to be suspended ketonuria occurred in two only, and, moreover, severe circulatory collapse occurred in the three cases in which ketonuria was not present. These findings do not correspond with those of Ström-Olsen, who, although analysing his results in a slightly different manner, makes it quite clear that he connects the occurrence of ketonuria with the occurrence of toxic complications, and regards it as the best indication of impending danger.

Mortality.

No cases of death were met with. This I attribute to the close attention to, and the careful preparation of, the charts, and also to the fact that I never hesitated to suspend treatment in any patient in whom toxic complications of the severity described in Section 4 had arisen. Not all authors publish a list of cases in which treatment had to be suspended, but Monnier (69), in 1936, reported 20% of 100 cases treated. This is exactly the proportion which occurred in this series. Ström-Olsen (5) had to suspend treatment in 4 out of 43 cases, but a further 4 patients had their treatment considerably modified as a result of severe complications.

Mode of Recovery.

Those patients who recovered fall into three groups : those whose recovery was apparent immediately following treatment ; those who began to recover

* Cf. Muller (2) who collected all mortality figures to 1925, equivalent to 5%.

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gradually ; and those whose recovery was characterized by an initial phase of recovery followed by a relapse and finally the gradual complete recovery. This last phenomenon of two phases in the recovery of the patient does not appear to have been mentioned in the literature, and it was only towards the end of the series that its occurrence gradually began to be recognized as a feature and noted in the case-records. In consequence an analysis of the frequency with which it is recorded would be misleading. It is recorded in three instances (W. 7, W. 9, W. 11), but in a subsequent review of the material I now realize that the same phenomenon took place in Cases W. 8 and W. 14.

6. Conclusions Regarding the Use of Insulin and Glucose.

In this research insulin and glucose were given with each dose of somnifaine as a routine. This technique was first advocated by the Cardiff workers, and especially by Ström-Olsen, who, as a result of his experience, came to the conclusion that " by the simultaneous administration of glucose and insulin to patients undergoing somnifaine narcosis, I have succeeded in rendering the treatment much safer through the elimination of ketosis and other symptoms, e.g., collapse and cardiac failure, which may be dependent upon the specific metabolic disturbance ". (Private communication quoted from M.D. Thesis, University of Wales, unpublished.) These conclusions of Ström-Olsen were made the subject-matter of two articles in the *Journal of Mental Science* (lxxix, p. 638, 1933, and lxxx, p. 658, 1934). These views were also upheld at the 1936 Annual Meeting of the Royal Medico-Psychological Association.

It appears then that as a result of the insulin-glucose technique the Cardiff workers claim to have buffered the continuous narcosis treatment against its main dangers. They further attribute these dangers directly to the effect of the barbiturate on carbohydrate metabolism, and they would appear to use the presence of ketone bodies in the urine as the best index of approaching toxic complications.

My experience was not in keeping with these views. In the first place I did not find that an examination of the urine for the presence of ketone bodies was of material aid in estimating the condition of the patient, or in warning me of cases who subsequently became ill ; and in the second place I met with quite serious cases of collapse in spite of the fact that I was employing insulin and glucose. It is difficult to say how these cases of collapse can be attributed to a metabolic disturbance, since in most cases they responded to the administration of fluids, coramine or cardiazol rapidly and effectively. The clinical appearance of the patients was that of circulatory collapse, and was invariably associated with a low blood-pressure. It would appear to be much more reasonable to attribute the majority of these cases of collapse to the action of the drug on the central nervous system, which we know to be the characteristic effect of the drug, and also to its action on the peripheral blood-vessels, which is also known to occur.

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This research, however, though demonstrating that the use of insulin and glucose does not deprive the treatment of its dangers, cannot claim to offer any direct proof as to whether or not insulin-glucose contributes in some partial way to rendering the treatment more safe. This would only be possible with a controlled series, but it is interesting to note that in this series of cases the treatment had to be suspended in 10 cases (20%), and this figure is the same as that given by the Zurich workers (69), who do not employ the insulin-glucose technique.

It is interesting to recall the fact that these conclusions are the same as those arrived at by Parfitt (22).

One piece of evidence which would appear to be difficult to fit in with Ström-Olsen's hypothesis, is the frequency with which serious complications, when they are met with, occur within the first six days, and especially on the fifth day. Were the toxic complications the result of a specific carbohydrate metabolism disorder, one would have expected this to show itself progressively, so that the majority of complications would have been met with towards the end of the treatment, e.g., the eighth, ninth or tenth day. Moreover, since the Cardiff workers in their hypothesis equate this specific carbohydrate metabolism disturbance to the narcotic action of the drug, one would have expected to meet the majority of these cases of complications in those patients who were exhibiting at the same time a pronounced degree of sleep. This was not my experience.

Finally, it is difficult to see why the question of habitus should have such a direct bearing on the appearance of toxic complications, if Ström-Olsen's hypothesis is correct.

All three considerations appear to me to argue in favour of the fact that toxic complications, when they occur (provided the treatment has not been employed in patients in obviously poor physical health and condition), are the results of an idiosyncrasy for the drug, and it was my experience that those patients who slept well under the somnifaine did not, as a rule, prove difficult cases. It was as if one could divide patients into two classes with regard to their behaviour towards the barbiturates, those who reacted characteristically to barbiturate administration by sleep, and those who reacted by a loss of cardiovascular tone, shown especially by a fall in blood-pressure. We should expect patients who have an idiosyncrasy towards the drug to exhibit this within the first few days, and it is in accordance with clinical fact that patients of asthenic habitus tend to have a poor cardio-vascular apparatus.

From recent discussion with Dr. Ström-Olsen, I gather he is still of opinion that the glucose-insulin method is of value. Similarly, D. N. Parfitt (22), although meeting with serious and alarming toxic complications, including 3 deaths out of 53 cases treated, nevertheless expresses the opinion that the treatment tended to be smoother in those patients receiving insulin-glucose than in those patients to whom somnifaine was given alone.

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A recent review (81, 82) of work in Moscow suggests that the Russian workers are favourably impressed with the value of insulin and glucose.

7. Discussion as to Mode of Action.

Various theories have been put forward by the different authors to explain the mode of action of the treatment. Some are essentially psychological (10, 11, 33) in conception, others more physiological (7, 30). It may be said in addition that whereas one group of theories refers the effect of the treatment directly to the pharmacological action of the drugs employed, another group of theories sees the main effect of the treatment as a secondary result of the narcosis; that is to say, an effect produced by the subsequent psychotherapy rendered possible by the preliminary use of narcotics. Finally, some authors have put forward suggestions embodying all the above conceptions (69).

Kläsi (7, 30), whose introduction of the treatment took place *ex hypothesi*, suggested that the result was due to the cutting of a vicious circle between motor restlessness and the increased psychic excitation resulting therefrom, which in turn produced further motor restlessness, i.e., his main conception was that schizophrenic excitation has a retroceptive action. In addition, he suggested that the feeling of helplessness produced by the treatment led to the foundation for further psychotherapeutic efforts.

Oberholzer (10, 11), put forward the suggestion that there was a loosening of affective tension, though, as Ström-Olsen (5) remarks: "With this theory it is a little difficult to explain the benefit which would occur in cases of mania".

Furrer (6) suggested that during the narcosis the ego is in abeyance, whereby dissociated constellations relieve themselves of their excessive libido, and link themselves into closer associated connection. As a result the hierarchy of the ego complex is more easily re-established after the discontinuation of the narcosis.

Muller (2, 50, 138) re-stated Kläsi's ideas, but in addition suggested a more direct pharmacological action on the brain-stem, "a pharmacological rearrangement of the vegetative nervous system", and pointed out that the leucocytosis, increase in blood sedimentation and the Arneth count which took place, were proofs that some such action on the vegetative centres occurred. In addition he pointed to the occurrence of cases in which immediate improvement took place following on the treatment, and in which no active psychotherapy had been instituted.

Monnier (69), having stated his belief that the psychotherapy employed is "an all-important factor", is, nevertheless, of opinion that there is a somatic factor at work. He draws attention to the effects due to the influence of the narcotics on the vegetative nervous centres and to the "hypersympathetico-tonics", who, prior to the treatment showed dilated, often spastic, pupils, with raised blood-pressure and tachycardia, but who, as a result of the

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treatment, achieve a normal balance: "Just these patients showed the best remissions". By contrast he mentions a class of patients who maintain these signs of hyper-sympatheticotonicity after the treatment, and who fail to improve subsequently. He suggests that these latter observations support the hypothesis of there being a psycho-physical disturbance as a basis of the psychoses, and discusses the possibility that the seat of the pathological disturbance may be the thalamus. It is the barbiturate component of the drugs employed which, in his opinion, acts particularly on this region.

Meerloo (1, 42) mentions the pathological evidence of Van der Horst, who found degenerative changes especially in the ganglion cells of the medulla, cerebellum and optic thalami in a patient who had died after taking 8 c.c. somnifaine. The work of Keeser (107*a*) and Pick and Molitor (117) has already been quoted. Most pathological reports mention the intense perivascular infiltration in all the organs, but do not single out the basal ganglia as showing these appearances in particular (Gruber and Roberts, 89, 90; Wyler, 14 Ström-Olsen, 5).

W. R. Hess (122) believes that the disturbance of the psychic functions in nervous or mental disorder appear as parallel symptoms to disturbance of other functions regulated by the vegetative nervous system. He believes that one of the most important aspects of the treatment is the restoration of the sleep rhythm, and suggests that sleep is a special symptom of the vegetative control of animal function, whose function is to protect the psyche against exhaustion.

Meerloo (1, 42) also suggests that there is a double factor at work in the cure, namely, a psychological and a somatic factor, and Lutz (46) is of a similar opinion. This author draws particular attention to the importance of the occurrence of amnesia following the treatment. This point is also stressed by Ström-Olsen (5), who states that: "I am therefore of the opinion that unless the patient falls into a moderately deep narcosis so as to obtain complete amnesia, full benefit is not being derived from the treatment".

Certain American authors have paid attention to the possible metabolic effect on the brain.

Loevenhart (108, 109) suggests that the treatment results in improved cellular oxidation, and Lorenz* also mentions this fact, but thinks that vascular dilatation may be a contributory factor here.

Bancroft (110, 111) and others have developed the hypothesis that mental disorder can be divided into two main groups, in which a disturbance of colloid organization is the essential morbid factor. They suggest that one group is due to colloid over-agglomeration, namely, manic-depressive illness and paranoid schizophrenia, and that the other group is due to colloid over-dispersion, viz., catatonic and hebephrenic schizophrenia, and involutional melancholia. Sodium amytal they suggest is a coagulating agent, and

* W. F. Lorenz, *Journ. Amer. Med. Assoc.*, 1929, xcii, p. 880.

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sodium thiocyanate a dispersing agent, and by applying these two drugs to the appropriate type of case they believe they may produce a therapeutic result.

Lang and Patterson (56) claim to have obtained clinical proof of the above theories, but Henderson (112) and Lucas maintain that pharmacological researches do not support the above views, and so the matter rests as being merely an interesting contribution to the theories underlying the nature of mental disorder, especially in relation to the treatment by means of narcotic drugs.

Meerloo (1), in addition to the suggestion that the treatment has a psychological effect in facilitating contact between patient and physician, and the suggestion that there is a specific barbiturate effect on the subcortical areas, also raises the interesting point as to the possibility that the sudden disintoxication is one of the factors at work ; that is to say, that the cure is, as it were, a cessation phenomenon of the treatment.

Hoff (99) believes that the barbiturates act by blocking the psychic vegetative pathway, and Magnus (68, 79) believes that a condition of ketosis results, which, by producing a state of relative dehydration of brain-cells, promotes freer oxygenation and produces cerebral irritability.

A. W. Hackfield (64) and K. Hoffmann (62) have both combined a sleep treatment with protein shock. The former author points out that though sedatives of the cortical group have proved successful, the most usually successful are those of the subcortical group, and concludes: "Hence the point of therapeutic attack must be on the central part of the vegetative nervous system". He then proceeds: "Various autonomic dysfunctions are usually reversed while the patients are under the influence of the narcosis, e.g., a hyperchlorhydric will show an achlorhydria and *vice versa*, and with recovery these functions again return to their physiological norm." He draws attention to Pavlov's (120) observation on his dogs, which showed that reflex reactions express themselves objectively through the medium of various autonomic functions, e.g., salivary secretion. Pavlov was able to induce a condition which might be described as an artificially induced neurosis, which resembled in some ways the affective syndrome. Pavlov cured this by a bromide narcosis over a period of ten days.

Finally, mention should be made of a recent article by M. Boss (123) from the Zurich school, in which an extremely interesting, if somewhat abstract theory is developed, employing psycho-analytical conceptions. This author appears to be the only one to subject patients who have undergone the cure to a systematic questionnaire in order to make an exhaustive psychological study of their condition during and after the sleep treatment. As a result of this inquiry he develops the conception of the "ego" being a functional psychic organ, for whose differentiation and maintenance a certain degree of mental energy is necessary. The function of sleep is to rest the forces which maintain

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the ego in consciousness, and though in healthy people this occurs satisfactorily, the mechanisms fails in patients suffering from schizophrenia.

If now we survey these various theories it will be seen that we have for consideration :

(1) The facilitation of psychotherapy which occurs as an indirect effect of the treatment, and the result in great part probably of the state of artificial invalidism.

(2) A somatic effect due to the pharmacological properties of the drug, which may be aided by the alleged tendency of the barbiturates to exert a specific action on the subcortical regions, and

(3) The psychodynamic effect suggested by Boss.

Before venturing to align myself with any one or any combination of these theories, it is perhaps permissible to record one or two phenomena which I think have a direct bearing on the points under consideration.

The first is that in my series certain of the cases did not receive any psychotherapy whatsoever, either from me or from the nursing staff, this being due to the prompt and rapid recovery following on the treatment. Amongst the cases which fall into this group should be mentioned Cases R. 18, W. 9 and W. 16. One can only describe these patients as emerging from their illness as if from a condition of normal sleep, and though in the case of W. 9 there was a slight relapse before her eventual complete recovery, nothing that can be described as psychotherapy, unless one is to extend the use of this term to the normal kindness of members of the nursing staff towards her, was employed.

These observations incline me to the belief that whatever other factors are at work, one is dealing in some cases anyhow, e.g., in those just mentioned, with a definite somatic effect which in turn is due to the pharmacological properties of the drugs.

That there is a very considerable alteration of brain function produced by the drugs, in addition to the phenomena of sleep, is shown by the existence of a psychotic phase, which has been referred to by only a few of the authors. This occurred very strikingly in the case of R. 1, who though both before and after his treatment conversing perfectly rationally, during his treatment became an actively hallucinated man with defective habits, and in general behaved like an advanced psychotic patient. There is also the phenomenon frequently referred to by the American authors, which I saw in only one case (not included in this series), in which a mute, catatonic schizophrenic will, during the treatment, become talkative and describe the hallucinations under which he is labouring. It seems to me, however, that the acceptance of the possible existence of a specific barbiturate effect rests on incomplete evidence. I have already pointed out that I can find nowhere in the literature a piece of research in which two series of patients have been subjected, the one to treatment by means of a cortical sedative, and the other to treatment by means of a subcortical sedative, and until this is done I cannot see how this point

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can be satisfactorily disposed of. Moreover, the Zurich workers (60, 61, 69) now use cloettal, in which, they maintain, the barbiturate component is only functionally speaking 50% of the total, and other authors, such as the Cardiff workers, are using progressively decreasing amounts of somnifaine and progressively increasing amounts of paraldehyde, and yet they continue to claim just as good results as had been formerly obtained by the sole use of a barbiturate, such as somnifaine. However, there are patients whose treatment is followed by an increased amenability to psychotherapy; this must be regarded as a very valuable effect. This class of patient as a rule dislikes being left alone, and will beg the nurses or the doctor to keep him company. This effect usually lasts for ten days to a fortnight, and then gradually subsides.

I inquired from a certain number of my patients what, in their opinion, was the effect of the treatment, and how they thought it had produced their cure. The results of this questionnaire were very disappointing, as most of the patients, beyond stating that it gave them good sleep and that they woke up very refreshed, feeling " different ", did not supply me with any data from which I could formulate any useful considerations. An exception to this was Case W. 9, who stated explicity that the main effect of the cure was the elimination of her ideas of reference.

In conclusion to this section I should like to refer briefly to the phenomenon mentioned in Section 5, in which five of my patients showed an initial period of remission from symptoms, followed by partial relapse, and then a final cure, the latent interval being in all cases about ten days, and being exactly comparable with the interval of freedom occurring in many of the patients who subsequently relapsed completely. In this latter group one could see the psychosis, once more taking a grip on the patient, with the patient powerless to do anything about it. The explanation of this phenomenon appears to me to be that in many forms of nervous disorder one is dealing with a somatic and a psychological disturbance. The sleep treatment temporarily abolishes both these disturbances, and enables a refreshed psyche to get control again of a mind in which, in many cases the somatic disturbance, though still going on, gradually subsides. It is for this reason that I am of opinion that there is nothing to be gained from shortening the patient's convalescence.

This hypothesis would also help to explain the condition of patients such as R. 21 and W. 13, who, though entirely changed as the result of the treatment, nevertheless remained in an irritable condition, which later commenced steadily to improve. I think that a relapse into the psychosis can occur similarly from a recrudescence of the somatic, or equally well from a recrudescence of the psychic factors. An illustration of the former instance is patient R. 6. This patient experienced a complete remission for fourteen days following on the treatment, whereupon his old symptoms gradually invaded his mind like some malignant growth, and for about two days he was aware of this and asked me what was happening. An illustration of the latter instance is patient W. 19.

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in whose case a similar complete remission occurred. The day following a rather emotional scene with her husband who had visited her, she commenced to relapse, working herself up into a frenzy of remorse and agitation over her infidelity.

The change of phase which sometimes occurs, in which a depressed patient emerges from a condition of depression into one of elation (e.g., Case W. 13), is surely evidence in favour of the treatment producing effects which have nothing to do with the psychotherapy employed.

My opinion is, therefore that the treatment operates by subduing two sets of morbid influences, a somatic and parallel psychological mechanisms. Provided both these are not of too great intensity the patient emerges from the treatment in a condition in which the mechanisms involved can commence working once more in a healthy manner. In cases in which either one or other of these influences is of relatively great intensity, the patient is in danger of a complete relapse. An intermediate group exists in whom the morbid influences attempt to reassert themselves, causing a relapse which lasts a little time, to be followed by a permanent recovery. I believe that the problem resembles the clinical problem occurring in the treatment by quinidine of auricular fibrillation associated with signs of decompensation. One checks a functional aberration of cardiac rhythm temporarily, and thereby enables the myocardium, if this is not in addition too seriously damaged, to re-establish efficient cardiac function. A seriously damaged myocardium, however, will relapse; the necessity of the patient to earn his living in a strenuous occupation will similarly result in a relapse. By analogy a damaged mechanism will result in relapse in the case of a mental illness; a troublesome psychic milieu will have the same effect.

8. Conclusions Regarding the Selection of Cases and Drugs to be Employed.

In this series of cases best results were obtained in the anxiety types and secondly in the depressive group. It is also clear that a far greater proportion of recoveries occurred in the "W" series than in the "R" series, which was to be expected. However, I feel that the case-histories of those patients who recovered make it reasonably certain that their recovery was the result of the treatment, and that my results are not open to the criticism that the treatment merely coincided with the period in which the patients would otherwise have got well.

The next finding of importance is that patients of pyknic habitus offer a good field for the treatment, and that asthenic patients are to be avoided unless other measures fail.

In general the various authors are in agreement concerning the type of

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schizophrenic patients who do best under narcosis, but there is much less evidence concerning the manic-depressive patients, and surprisingly little concerning the psychoneurotic patients.

Kläsi (7, 30) advised against giving acute cases of schizophrenia the treatment, taking the view that the illness had a natural course in which the tendency to chronicity must be prevented by the treatment. He suggested the following groups:

1. Agitated, depressed and restless types.
2. Antagonistic, negativistic and autistic types.
3. Acute hallucinatory episodes.
4. Cases exhibiting stereotypies.
5. Dispirited, undecided, indifferent types.
6. Hysterical types.

And finally all cases who solicit the treatment and realize that they are ill.

Müller (2), Oberholzer (10) and Lutz (46) endorsed these findings, and added that the more nearly the illness approached the true manic-depressive type the better the results. Lutz, however, criticized the value of the treatment in the third group with acute hallucinatory episodes.

Monnier (69) states that the best results are to be obtained in chronic cycloid schizophrenics, manic schizophrenics in the subacute stage, psychogenic excitable hebephrenics and schizoid psychopaths. This author believes that a very short period of four days' treatment suffices for the psychogenic excitable episodes of schizophrenia. This statement accords with the results obtained in Case W. 9.

Cloetta (60) and Maier (61) recommended the treatment for:

1. Acute attacks of catatonia and hebephrenic forms in their subacute stage, especially those who seem unable to get into contact with their fellows. Also cases who are developing automatisms, excitable movements, negativisms and dissociation, with or without delusions.

2. Cases showing pronounced manic or depressive components, provided chronic catatonic symptoms do not predominate.

3. All acute episodes in schizophrenic illness.

4. Chronic excitable catatonics, whose excitability is often much diminished by the treatment.

It will be seen that the majority of the Zurich case material consisted of schizophrenics.

The American literature (49, 51, 55, 57, 58, 76, 77), apart from Palmer and Paine (58*a*), shows that clinicians in the U.S.A. have directed their attention more to much modified techniques, so that their results are not strictly comparable. This consists essentially of giving the patients doses of sodium amytal daily, and utilizing the patients' subsequent remarks as a basis for making better contact with him. Palmer and Paine obtained their best results in manic-depressive illness.

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Meerloo (1), whose experience covers 500 cases, emphasizes the impossibility of predicting which cases are likely to benefit most, and in addition expresses the opinion that it is not known which phase of the various syndromes constitute the correct point of therapeutic attack. To the Cardiff workers, and especially to Ström-Olsen, belongs the credit of emphasizing the value of the treatment in manic-depressive illness and in the psychoneuroses. Hennelly (44), whose figures include and whose results maintained Ström-Olsen's original conclusions, shows a recovery-rate of 36·8%, and he concludes that "in 30% cases of manic-depressive psychosis the illness can be cut short in two to three weeks by the treatment". Ström-Olsen's figures relating to the psychoneuroses showed 8 recoveries out of 13 patients. This figure includes 5 anxiety types out of 7 treated. MacNiven (43) reported on 40 mixed cases to a meeting of the Royal Medico-Psychological Association (*Journ. Ment. Sci.*, July, 1936) and found the most favourable field in his group of melancholia, and in addition obtained permanent improvement in the majority of his cases of mania.

The Twenty-second Annual Report (45) of the Board of Control shows that in eight mental hospitals the treatment had received a trial during 1935, and in six of these hospitals the reports are favourable. In only one instance, however, was the number of cases treated greater than twenty patients. The experience, on the whole, appears to be in keeping with the figures obtained in this research. These reports include one from Parfitt. This author originally published (22) a series of cases and obtained good results in his manic-depressive series. In the report referred to above he appears to be using the treatment a little less than he had done formerly.

Before leaving the discussion regarding the selection of cases to be given the treatment, there is one opinion I should like to express, which, so far I have not seen mentioned in the literature, namely, that those patients in whom an element of distressed perplexity (cf. 125) is predominant, appear to me to afford a very favourable field. This was particularly noticeable in some of my depressive patients who, prior to the treatment, complained that they felt as though their brains were in a constant state of turmoil. It is patients of this class who so frequently find themselves unable to think out the necessity for treatment, or in so doing further increase their general sense of worry. It seems to me a virtue to be able to avoid certification of this class of patient, and they very frequently do not settle down to treatment along voluntary lines. Continuous narcosis, by diminishing even though not abolishing entirely this worry, enables the necessity for treatment to be comprehended and accepted by the patient, so that it is possible to treat them in a manner which would otherwise be impossible.

Regarding the choice of drugs to be employed, it is clear that the Zurich workers having tried somnifaine (7, 30), dial (46, 50) and luminal (10, 46), have abandoned the exclusive use of the barbiturate and now employ cloettal (60, 61, 69). In the U.S.A. sodium amytal is used almost exclusively in connection

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with narcotic treatments. In this country somnifaine has been the barbiturate employed.

My opinion is that the exclusive use of somnifaine constitutes an unjustifiable threat to the patient's life, and moreover limits the field of application of the treatment and at the same time renders more difficult the therapeutic object, which is to obtain the maximum amount of sleep compatible with the patient's safety. I do not know that any scientifically indisputable clinical evidence has been brought forward to prove the existence of a specific barbiturate effect on mental illness. I think that the continuation of just as high a proportion of recoveries by the use of cloettal at Zurich, and the recoveries obtained in this series by the use of paraldehyde together with a barbiturate, can be considered as partial evidence against this hypothesis. Nevertheless I consider the barbiturates to be valuable therapeutic aids in procuring and maintaining sleep.

In my experience somnifaine is both safe and effective if employed together with paraldehyde, but I have no experience of the other barbiturates. A comparative study of the value of the different barbiturates along the lines essayed by P. Hoch (65) as applied to this form of treatment, would be most valuable.

Perhaps one of the most interesting variations in technique is that of Segerath and Wember (8), who give a battery of drugs according to a scheme commencing each day at 7 a.m. and finishing at 8 p.m. This method is attractive in that accumulation of any component is entirely avoided, and possesses the not altogether hypothetical virtue that the combination of narcotics having different points of selective action ensure a more thorough narcotization of the brain.

My conclusions concerning the insulin-glucose technique are contained in Section 6.

Concerning the choice of restoratives and antidotes: the various authors have advocated cardiazol, coramine, ephedrine, ephetonin, caffeine, strychnine, camphor and digalen. In this research I used cardiazol and coramine, and found them rapid in action and effective, and safe to use. Occasionally I used caffeine to promote diuresis, and very occasionally a small dose of bismuth and belladonna in cases with nausea. I do not believe that any of these drugs constitute as sure a prophylactic as the administration of adequate fluids, which should not be less than three pints in twenty-four hours.

9. Conclusions Regarding the Value of Continuous Narcosis as a Method of Treatment.

The chief conclusion arrived at in this paper which I hope is justified by the results, is that continuous narcosis is a valuable method of treatment with a wide range of usefulness for patients suffering from both the milder and

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more severe forms of mental disorder, and I regard its value as sufficient to justify its use in any psychiatric clinic. This conclusion is still the subject of much controversy, and whereas leading psychiatrists, such as Lange, Mactie Campbell, and N. D. Lewis, in addition to at least one of the leading German psychiatric clinics,* do not regard it as at all useful, others, such as the Zurich and the Cardiff workers are loud in its praise.

My own finding is that in a series of cases including representatives of the chief varieties of mental illness, the treatment is capable of producing an all-round recovery-rate of approximately 33⅓%, and although these recoveries take place in the main in patients in whom a favourable prognosis is otherwise to be expected, they take place as a direct result of the treatment as far as the speed of recovery is concerned. Moreover, the treatment, in addition to hastening and promoting recovery, does this in a manner which is agreeable to the patients, and in consequence is much appreciated by them.

In consequence I find myself in entire disagreement with opinions such as that of Gillespie, to the effect that the treatment is more of adminstrative than of therapeutic value.

The treatment is not without its dangers, and serious complications can be met with which cannot be foreseen or entirely avoided, even with the most careful attention to the details of case management or the administration of insulin-glucose. These dangers, however, when they occur, can be dealt with rapidly and effectively, so that in my opinion the risks are more than justified by the results which can be obtained. I therefore entirely disagree with those authors who regard the dangers of the treatment as a contra-indication to its employment. Provided adequate caution is displayed with regard to the choice of suitable case material, and careful attention is paid to the appearance of toxic complications indicating the necessity of stopping the treatment, no fatalities need occur, and in this connection I regard the routine preparation of clinical charts, together with the administration of adequate fluids as the most important safeguards in case management. I am also of the opinion that the quality of the respirations and the state of the blood-pressure are the most valuable measures of the patient's condition during treatment. I therefore disagree with the Cardiff workers, who appear to contend that ketosis is the complication to be most feared, and it is not my experience that the insulin-glucose technique protects against the appearance of toxic complications to anything like the extent claimed by these workers.

I do not believe that the beneficial effects of the treatment can be explained on psychological lines entirely, and I have brought forward evidence which I think supports the view that there is both a somatic and a psychological effect at work, and in the former instance I have also brought forward evidence against the view that the major effect of the treatment is due to a specific action of the barbiturates.

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Finally, I feel justified in claiming that the technique and methods described which combine the use of a barbiturate with the use of paraldehyde, constitute a reasonably safe and effective technique in achieving the therapeutic object, namely, that of putting the patient to sleep for an average of eighteen to twenty-two hours daily for a period of eight to ten consecutive days.

Summary.

(1) The treatment is of value in a psychiatric clinic.

(2) In a mixed series of cases it yields approximately 33⅓% recoveries. These recoveries occur especially in forms of mental illness which would otherwise have had a good prognosis.

(3) These recoveries are due to the treatment.

(4) The treatment is not without danger, but the risk can be effectively safeguarded and fatalities need not occur.

(5) The claims put forward on behalf of the insulin-glucose technique by Ström-Olsen and others are not substantiated in this research.

(6) The effect of the treatment is probably due to both somatic and psychological factors, and as regards the somatic factor a specific barbiturate effect does not appear to be very marked, if indeed it occurs.

(7) The combination of somnifaine with paraldehyde constitutes a reasonably safe and effective technique.

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EPILEPTIC REACTIONS: AN ATTEMPT AT CLASSIFICATION.

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THE NECESSITY FOR REVISED CLASSIFICATION.

The subject of this essay has almost the blessings of antiquity. The increase in the knowledge of the ætiology of epilepsy makes the revision of its classification very necessary. It has been well said that complexity of nomenclature implies poverty of knowledge. This is conspicuously true of one branch, in particular, of medicine, where the desire for classification has outrun the accumulation of data. It is not so with epilepsy, where increased knowledge of causation necessitates a revision of the scheme of classification.

It is not proposed to deal at any length with previous systems, except to mention the broad subdivision of the condition into idiopathic and non-idiopathic. The addition of knowledge in recent years has led to the gradual erosion of the basis of the idiopathic conception, which is now so closely approximating to vanishing point that it can conveniently be discarded. Where the term "idiopathic" is used, the subsequent history reveals how surely the fortress of negation must finally capitulate. Many cases previously described as idiopathic have since become classifiable as biochemical catastrophes, or glandular dyscrasias. The great argument against the employment of the term "idiopathic" is, however, that it included many cases of gross developmental non-epileptic abnormality, so that many cases of oligophrenia, or of that disease included under the unfortunate term of "epilepsy with insanity", present obvious degenerative features which the term "idiopathic" tends to minimize. A further important objection is that this term was used to include manifestly dissimilar clinical categories. To quote a crude example, cases of oligophrenia and of epilepsy with insanity, as well as those of non-deteriorating varieties of epilepsy where the causation was indefinable, were, in employing the above term, credited with a similar ætiology.

POSSIBLE CLASSIFICATIONS.

In endeavouring to discover appropriate classifications for epileptic conditions it is necessary to remember two inextricably connected factors: firstly, the inherent instability of the nervous system, and secondly, the irritating factor which precipitates the convulsion. Were there any clean-cut distinction of cases where one or other factor could be regarded as mainly causative, a

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broad classification would naturally suggest itself. But while much macroscopic and microscopic evidence of lesions and cerebral lesions is available in cases of epilepsy, these are also found where there are no convulsions. A similar state of affairs exists with such chemical and metabolic disturbances which act as convulsants. In some cases the conditions present cause convulsions; in others they do not.

Where two or three similar types of disease exist, and provide troublesome problems in their classification, it is a point of general agreement that the soundest basis for classification is the pathological one. In epilepsy, however, we are dealing with a condition which is erroneously described as a disease, and which is best regarded as a particular type of response. Any attempt at pathological classification arouses mathematical problems too complex even for this age of convenient formulæ.

It is well to consider the effects of classification on a pathological basis, since this latter is a proceeding of much scientific justification. In the first place we would find it necessary to consider those cases the origins of which were primarily in the nervous system. These would comprise the catastrophes of parturition, meningeal and cerebral hæmorrhages and thromboses, Little's disease, the allied paraplegias, the various varieties of meningitis and polioencephalitis, not to mention intra-uterine infections and cataclysms. Congenital developmental aberrations, the absence, or malformation, of necessary areas of cerebral tissue, or, in the absence of such dramatic evidence of insufficiency, a paucity or malformation of cells, and even an aberration of cellular function unrecognizable by any known present methods of investigation, would all have to be considered in this connection. When we take into account, too, those cases of epilepsy in which the causation is less manifestly infantile, even allowing for the fact that most research ultimately points to these factors being innate, the ætiological variability becomes even more confusing—so complex indeed that some classification as to the system at fault is necessary even before we can begin the consideration of them. To this end perhaps it is best to refer to these epilepsies under such by no means exhaustive headings as "glandular", "allergic", "biochemical" and "miscellaneous". Study of any of these subgroups will reveal manifold further subdivisions, until all we have achieved is to construct a veritable maze of ætiology.

So far as the glandular sub-group is concerned, the possibilities of still further ætiological subdivision are best indicated, not by dealing singly with the different glands, but by the mere statement that no gland has escaped scrutiny in this connection. A stringently scientific classification as to causation would therefore involve an enumeration of all the various gland dyscrasias under this one particular sub-heading. A classification as to an allergic causation would embody not only the elucidation of the various factors which might be held responsible, but also an inquiry into the justification

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for adopting the allergic view of causation. This is by no means clearly established. Such evidence as is produced by Ward and Patterson for that type of epileptic who is able to pursue a livelihood has less application in more institutionalized cases. Evidence as to the existence of the usual criteria of allergic states, e.g., eosinophilia, hæmoclastic crisis, etc., is most conflicting in the case of the different epilepsies. Standard text-books often refer to eosinophilia, for instance, as though it were a constant finding. The abundance of literature proves that this is not by any means the case. My own experience points to there being no increase of eosinophil cells (Guirdham and Pettit, 1936*a*). With regard to the hæmoclastic crisis the evidence for this is amazingly contradictory. In the hæmoclastic crisis a functional incapacity, possibly transient, of the liver is mooted, and several observers have strongly urged the existence of this incapacity. Even allowing for the fact that the lævulose tolerance test is probably by no means an ideal test of liver function, it is of interest that, in an investigation conducted in conjunction with another worker, the findings indicated a functional capacity greater than normal on the part of the epileptics (Guirdham and Pettit, 1936*b*).

Where the desire for classification involves the "simple ideal of quantity before quality," the varieties of epilepsy included in the biochemical category should provide propitious material. There are the ketogenic varieties, the epilepsies engendered by hyperpnœa, and a whole host of others. It is in that blessed category we label as "miscellaneous" that the statistician finds his maximal expression. Here we range from those epilepsies produced by peculiarities in the coagulating properties of blood to Reed's almost ideal conception of the *Bacillus epilepticus*.

It must be understood that the immediately foregoing remarks embody no ideal of a pathological classification such as would be approved by the author. He has merely mentioned some of the groups which might be considered in such a scheme, and indicated the difficulties inseparable from such a course of action. The first defect of such a system is its unwieldiness. Classification is more than a prodigious vomit of labels. It involves the abstraction of totalistic conceptions from a superfluity of details. A disease itself is, relatively speaking, a totalist conception abstracted from a mass of symptomatic detail. It would appear to be the function of the diagnostician to construct, from the mass of epilepsies, disease entities analogous to those recognized elsewhere in medicine. Extensive classification is a symbol of insecurity. It is the key to a conspicuous lack of data. It is seen at its worst in certain of the more primitive spheres of psychiatry, where a few barely known and loosely connected facts are grouped together as new disease entities, and sent forth into the world in the gala dress of purposeless nomenclature. This tendency to classify epilepsy to the last decimal point is due to the mistaken conception of it as a disease. Epilepsy is a peculiarity of response, determined by a vastly variant category of explosives and, this being the case, a purely

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ætiological classification is merely the enumeration of so many different varieties of switch when what we are investigating is the nature of the current. The important point is that, within the vast area included in the confines of the term "epilepsy", there are certain disease entities to be recognized, and that these tend to be hidden by the obfuscation due to this over-labelling of causative factors.

Difficulties in Classification.

From the strictly purist standpoint there is much to be urged against an exclusively clinical classification of types. It seems to me necessary in the case of the epilepsies because here ætiological conditions have a twofold significance. We have to consider to what extent pathological factors are responsible for the convulsive tendency, and to what extent they tend to give rise to any particular disease entity. In the present state of knowledge it seems impossible to separate these two factors, so that clinical considerations may be assumed to be as reliable as any.

We have been concerned chiefly with an examination of the precipitating factors. Does the second feature in epilepsy, the fundamental irritability and instability of the nervous system, afford us any more reliable means of classification? I do not think such an attempt at classification should be made. To describe different varieties of epilepsy according to the area of the brain involved is no more logical than to regard cerebral monoplegia as an entirely different disease entity from cerebral paraplegia, merely because the incidence in the two cases is on different parts of the brain, or on the same area but to different extents. No classification as to degrees of irritability is possible. When we learn the threshold values for stimulants in nervous disorders we shall, perhaps, be approaching the golden age of medicine, but indubitably that day is not yet.

It might be argued that, even though a fundamentally clinical classification be contemplated, it is essential that each separated category should be accompanied by its appropriate pathological picture. But, despite the ubiquitousness, the variability in the lines of attack, shown in the growing investigations of the epileptic state, no clear-cut pathological picture has been found in association with any particular physical state. The variabilities in coagulation rate, in protein sensitivity, etc., apply to very heterogeneous groups of individuals. The great trouble with epilepsy is that no organ, not even the brain, so far as available methods permit, has been found invariably at fault, and that, in this latter case, the fault has often lacked definite location. Of the less reputable organs, perhaps the liver and the glands have been most indicted. The general findings as to a hypothetical inefficiency on the part of the former have specified this inadequacy of function as applying to no particular type of epileptic.

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The Necessity for Clinical Classification.

For these reasons the scheme of classification on which one is embarking is largely conducted on a clinical basis. It must be borne in mind that the writer is not so much seeking mathematical niceties of calculation as wishing to describe definite disease entities as they occur within the definition of epilepsy. While it would be ideal to deal with the different disease entities one proposes to describe in some developmental order, or according to the degree of mental impairment involved, it is a matter of necessity to describe first the morbid conditions whose manifestations are the most striking.

Progressive Epileptic Deterioration.

This is a disease the physical signs of which are variable but which involve the occurrence, in some form or other, of epileptic manifestations. The characteristic feature is the peculiar psychological state, so exhaustively described by Pierce Clark (1912, 1914, 1915, 1926, 1933) and McCurdy (1916, 1925). Were one limited in one's expression to the most salient features of the disease, it would be best to describe it as a state of mind intersected perpetually by a peculiar form of egoism, which, gaining crude objective expression in its early stages, results finally in hypochondriacal preoccupations accompanied by singularly little affective disturbance. Egoism alone would seem to be a singularly insecure method of diagnosis, and of course one does not rely solely on it, but the form of egoism is peculiar in that it is without even intangible justification. The egoism of the paranoid patient has often supplementary delusions. It is easily understood where a patient believes himself to be a king. Where such manifestations are present the activities of the delusional patient have a point and a superficially rational justification in the elevation of his mien and the self-assertive serenity he displays to the world around him. The main distinction is that whereas in the paranoid the egoism is continually and to some degree logically purposive, in the epileptic it is more episodic and always more blindly purposive. The paranoid tends, by the elasticity of his mental processes, to mould the environment to his purposes. The epileptic's egoistical outbursts are more definitely provoked by his environment. In saying that the egoism of the paranoid is continually displayed, one wishes it to be realized clearly that with the epileptic too, egoism, estimated over a period of years, is a constantly present factor, but that its periods of most evident eruption are more episodic than, say, in the smiling, invulnerable, and exasperating serenity of certain types of paranoid. Further distinguishing features are, of course, the incidence of convulsions in the epileptic and the absence of such destructive deterioration in the paranoid.

The other most characteristic feature of this progressive epileptic deterioration is the peculiar variability in temperament seen in no other disease.

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Alterations of mood are present in most mental conditions, but the typical variation in this condition is not so much of mood, as an amazing, almost quantitative variability in the expressiveness of the temperament as a whole. In the intervals between his more dramatic outbursts the epileptic betrays an inordinate degree of perceptive obtuseness and emotional apathy, surprising in patients who, whatever may be thought by casual observers biased by the crude melodrama of the epileptic convulsive phenomena, remain among the most normal of patients in mental hospitals where, by normal, one refers to the simpler ratiocinative spheres. In these "turgid" or "viscid" periods, as certain authors have described them, the patient appears to be existing in a state of suppressed charge, inevitably to be dissipated in some furor totally disproportionate to its exciting stimulus, or in a series of convulsions.

It is unnecessary to describe too fully the whole picture of these cases. The mental symptoms as such are embodied in any reputable description of the epileptic temperament, though too many theses on the subject appear to regard symptoms like irritability as peculiar to the epileptic and unknown among the general public. The point to emphasize is that the association of the epileptic temperament, with epileptiform manifestations, accompanied by a gradual progress towards a distinctive variety of deterioration, constitutes a constellation of symptoms which ought to be considered as a definite disease.

Epileptic Retardation.

It is necessary to deal now with another condition with which progressive epileptic deterioration ought not to be confused. This is what I propose to call "epileptic retardation". This condition may be confused with progressive epileptic deterioration in certain phases of the latter's development, a state of affairs probably contributed to by the fact that both types of case are found together in institutions. Epileptic retardation is, however, a condition whose early history is vastly different from that of progressive epileptic deterioration. In the former there has been present oligophrenia of some form or other from an early age. The name chosen for the disease is deliberately selected so as to include both truly congenital cases, and those where the morbid condition has been acquired later, though still at an early age. Much literature has been devoted to the distinction between epileptic oligophrenia and oligophrenia with epilepsy. Much of this writing is, in the present state of knowledge, somewhat superfluous from the point of view of classification, since it is impossible to assert to what extent convulsions are conducive to deterioration, or symptomatic of a particular variety of it. A point which should be emphasized is that conditions accompanied by microscopic pathological lesions of the cerebrum, as in nodular sclerosis, and by gross developmental anomalies, such as the absence or maldevelopment of a part of the brain normally important in humans, are best excluded from notice. These

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conditions are often accompanied by well-developed degenerative stigmata. The cases described under epileptic retardation are not characterized by the development of such features to any extent greater than that shown in those forms of oligophrenia where physical malformations are less specifically marked. Except in cases of the marked oligophrenia of the idiot and lower imbecile category, the incidence of degenerative stigmata in aments with epilepsy has been grossly exaggerated. Such classes as these should be excluded from description under epileptic retardation, since general developmental peculiarities consistent with gross brain lesions are usually manifest in them. In the case of epileptic retardation it is not considered advisable to include cerebral pathological conditions beyond the cellular deficiencies and peculiarities characteristic of oligophrenes other than the most gross cases, and beyond the common under-development of the higher cortical layers. It is very necessary to emphasize this point, since it is probably wisest, in this discussion, to exclude from notice cases where convulsions are due to a tangible, grossly mechanical irritant such as a tumour formation. The inclusion of such cases in discussions of epilepsy is deleterious. Its logical end is the inclusion, in kinship with epilepsy, of conditions like cerebral hæmorrhage. The argument that any condition with any epileptiform manifestations ought to be classified with the epilepsies is not a very sound one. A gross irritant like a tumour, or the escape of blood from an artery, is likely to arouse convulsive effects in tissues not inherently predisposed to such phenomena. It must be remembered that epilepsy should describe an innate tendency to convulse.

I do not think it can be said that there is any particular characteristic of the oligophrenia in epileptic retardation. It may be argued that, this being so, the description as a disease entity of the symptom-complex which included epileptiform seizures and oligophrenia is fallacious. This does not take account of the fact that we are deliberately excluding from notice cases where gross constant precipitants of convulsive phenomena, i.e., tumours, are present, which therefore helps to demarcate epileptic retardation more clearly.

Much of the objection to this disease conception of epileptic retardation arises from dwelling too much on the great diversity of cases where oligophrenia and epilepsy are associated. It may be held that to sort a single clinical entity from such a heterogeneous collection of types is almost too idealistic an endeavour. The rejection of cases with obvious mechanical precipitating causes, i.e., tumours, chronic meningitis, etc., clarifies the perspective considerably. The remaining field is also restricted if we are prepared to review our attitude towards developmental peculiarities in epilepsy. It is constantly assumed that gross developmental stigmata are markedly present in the epilepsies. This has not been my experience. I submit that many of these cases where gross stigmata are present are associated with gross cerebral and central nervous lesions, and abiotrophic peculiarities which in

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themselves tend to be explanatory of the convulsive phenomena. It may be argued that, if we are dealing with epilepsies as a whole, why should we exclude from consideration such tangible precipitants? The answer is that in most epilepsies the purposes of science are best served by studying what Notkin calls that "inherent tendency to convulse", and that in these cases of gross cerebral lesion no inherent tendency would appear to be relatively necessary, in view of such obviously precipitating factors. It may also be argued that these cases should be placed in a separate category. There is no reason why this should not be done, and where eager enumeration is the goal of the investigator, such a proceeding should be a source of considerable pleasure. The point will long be debated whether the oligophrenia is a consequence of the convulsions or an association with its cause. Be that as it may, it seems short-sighted not to recognize as an entity a useful conception like epileptic retardation, merely because it is impossible to determine whether the oligophrenia or the epileptiform condition is the prepotent determining consideration.

Distinction between Progressive Epileptic Deterioration and Epileptic Retardation.

There is no doubt that certain stages of progressive epileptic deterioration are closely comparable with epileptic retardation, but this would apply only when the deteriorative factor in epilepsy is becoming profound. There are, however, three distinguishing factors applicable at any stage. Firstly, the existence of the epileptic temperament. The point has not been sufficiently emphasized that this is shown in its maximal development in that class of patient at present described under the undesirable heading of "epilepsy with insanity", and to a much smaller extent in the more ambulant and yet unoccupied cases of epilepsy, who, in the more favoured classes, gravitate to the neurologist's consulting-room. The salient fact is that the epileptic temperament is little developed in those cases which have been much more unmistakably oligophrenic at their inception.

The second point is the previously higher intellectual endowment in the case of progressive epileptic deterioration. This may seem a startling statement to those whose conception of the epileptic is derived from his condition when conspicuously advanced, but, as Pierce Clarke (1915, 1933) and McCurdy (1916) have shown, a very striking feature of the epileptic psychology is its absence of interest. When it is possible to arouse this latter faculty the degree of latent intelligence is greater than might have been supposed.

Finally, we have the question of the disintegrating effect of the convulsions. It is a well-known observation that the effect of cessation of the fits in cases similar to those I propose to call "progressive epileptic deterioration" is very often the reverse of beneficial. This does not imply that in some cases the

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patients are not benefited. On the other hand, where we are dealing with epileptic retardation, the diminution in the number of the fits appears more commonly to have some beneficial effect.

Essential Epilepsy.

The next category we will deal with is that for which the name "essential epilepsy" is suggested. By this we intend to imply those cases where the convulsive element is the most salient feature of the disease, and where the element of psychological degeneration is not well marked. These cases comprise the more ambulant varieties of epilepsy. Many are enabled to pursue their livelihood with relatively little interruption, and with no wholesale impairment of intellectual functions. It might be argued that these cases, and those of progressive epileptic deterioration, essentially comprise the same disease process, and represent different degrees of it. It might be thought that the deleterious effect of the convulsions, due to a more established degenerative tendency, is greater in the case of the progressively deteriorating cases. Such a view embodies two main fallacies. In the first case it has never been proved that the convulsions alone are deteriorating influences. The accumulation of evidence, except in the more oligophrene varieties of epilepsy, suggests that the reverse is true. In the second place, if this tentative, hypothetical view be true, why should the primary distinguishing feature of progressive epileptic deterioration be always a peculiar and unmistakable cast of psychology, present in many cases even before the onset of convulsions?

There is, of course, no distinguishing feature about the character of the convulsions, or *petit mal* manifestations, in this type of epilepsy, except in the nature of the precipitating circumstances which we will deal with later. There is no clean-cut mental picture associated with essential epilepsy. In some cases a degree of retardation is present. Scholastic backwardness is not uncommon. In others, anxiety states are present, as though, in the words of Thornton Wilder, the patient anticipated "those moments which separated him from other people". In others a pathetic, dependable state ensues. In some the intellectual state is normal, in others subnormal. Others, like Cæsar and Mahomet, are conspicuously gifted. There is no uniformly occurring psychological condition accompanying epilepsy of this type. It has to be admitted, too, that a certain proportion of ambulant epileptics, of the variety living at home a supervised existence, but not engaged in an active, bread-winning occupation, show signs of the epileptic temperament. Apart from the fact that imperceptible gradations from one adjacent category to another are inevitable in all forms of illness, it is probably wiser to classify such cases as those of progressive epileptic deterioration. Such signs of the epileptic temperament as appear are likely to lead to deterioration of some degree, and at any rate, the very possession of an epileptic temperament in whatever

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degree of development it exists is, of itself, an incipient deterioration, since the egoism and constriction of interest which constitute the main motivating factors of deterioration are among the chief foundation-stones of the epileptic temperament.

Aids to the Diagnosis of Essential Epilepsy.

It must be admitted that this category of essential epilepsy is not nearly so clearly demarcated as that of progressive epileptic deterioration. It includes conditions the precipitating mainsprings of which are vastly dissimilar. This objection must apply to the epilepsies as a whole, though it is more noticeable in this condition, where the multiplicity of precipitating causes is greater. The greatest proportion of protein-sensitive epileptics occurs, as has been pointed out by Wallis and Nicol (1923), in an ambulant type of case quite distinct from cases of progressive epileptic deterioration. The same seems to apply to epileptics best responding to snake venom, i.e., a type of case in which either protein sensitivity or peculiarities of blood coagulation are most potent as *causes* of the seizures. I cannot but think that the majority of cases responding best to such measures as the ketogenic diet are cases of essential epilepsy, rather than of progressive epileptic deterioration. It is impossible to be dogmatic about this, because of the very considerable lack of specificity in describing the epileptic subjects of these experiments. There is, however, certain presumptive evidence that the above supposition is correct, in that most experiments with ameliorative measures which have had the best results in dealing with epilepsies, have been performed on the ambulant rather than the institutional case. This leads us to two further interesting factors, useful in demarcating the different epilepsies. Firstly, as a general rule, the most recoverable varieties are those least contaminated with the epileptic temperament. It is, in fact, this latter possession, with its intrinsic possibilities towards disintegration, which demarcates most saliently the clearest epileptic type. The second point is that there is strong evidence that there is a greater variety of obvious causative factors in essential epilepsy than in progressive epileptic deterioration. In the latter, indeed, what we might call gross pathological (excluding *psycho*pathological factors), explanatory factors are far less evident than might be expected from perusing some of the old-fashioned dazzlingly ubiquitous chronicles of epileptic ætiology. It is difficult to say dogmatically that these gross and tangible exciting factors are less manifest in progressive epileptic deterioration than in epileptic retardation and essential epilepsy, but it may well be that this may subsequently be found to be the case. To take, as an example, one recognized precipitant of epilepsy, i.e., glandular dysfunction. It is surprising how few cases of progressive epileptic deterioration present signs of glandular dysfunction. This applies of course to pituitary dysfunction, which plays at least a recognizable part in the genesis

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of some epilepsies. Careful observation of available facts will, I think, lead to the conclusion that in so far as we are dealing with the commoner excitants of the central nervous system, i.e., glandular dysfunction, metabolic dyscrasias, etc., these are less evident in progressive epileptic deterioration than in essential epilepsy. In the former case the disease does seem to rest more securely in the personality of the individual, and in the abnormally convulsive tendency of his nervous system, than in the presence of physical excitants. He, more than the essential epileptic, provides his own stimuli. This does not imply that subsequent, more minute research may not find an adequate and tangible physical excitant in the case of progressive epileptic deterioration. One cannot see, if the tendency of science at the present day be any indication, how it can fail to do so. Yet one is justified in emphasizing strongly that progressive epileptic deterioration is more essentially what we might call an intrinsically psychological disease than is essential epilepsy. A further point of great importance is to study the effects of such recognized types of treatment of epilepsy as are based on the presence of known pathological peculiarities, e.g., the ketogenic diet as a measure combating the alkali-tending epileptic, the desensitization of epileptics abnormally reacting to proteins and other substances with an allergic reaction in particular cases, the response of Spangler's cases to snake venom. It will be found that the results in these cases are mostly derived from the ambulant, non-institutional epileptic. The latter still benefits most by the sedation of his cerebrum. Making allowance for the fact that all institutional epileptics are not by any means cases of progressive epileptic deterioration, I cannot but feel that it will later be proved that the therapeutic attack on these last-mentioned cases should be made on the epileptic personality and the strongly marked tendency to convulsive reactions, and that in the case of essential epilepsy the removal of the excitants will be the most rational mode of procedure. This does not, of course, mean to decry in any way that natural tendency towards convulsions which, by our very definition of the condition, is present in epilepsy of any variety. It is only that in the case of progressive epileptic deterioration we appear to be dealing with a condition in which the tendency to convulsions is very strong, and appears to be more fundamentally engendered in the mental and nervous fabric of the individual than in some other cases which rely on chemical and glandular excitants to produce their most dramatic effects.

Epileptic Abreaction.

So far we have not discussed psychological factors as immediate excitants of epileptic convulsions. In dealing with the epileptic temperament we have merely discussed the psychological foundations of a certain condition, i.e., determining, rather than exciting, psychological causes. It is these latter which are of considerable importance in this present connection, since we are

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to describe a type of epilepsy mostly precipitated by psychological factors, and which has, furthermore, a greater content of psychological manifestations than any type we have hitherto described.

Our choice of a name for this variety of epilepsy is rendered extremely difficult by the enormous confusion of terminology there has been in describing conditions with epileptiform manifestations and also with psychological signs, perhaps particularly those of a psychoneurotic nature. This new category we propose includes cases previously referred to under the heading of "hysteria with epileptiform convulsions, hystero-epilepsy, and psychogenic epilepsy". These terms have no more than a general indicative value, on account of the considerable confusion as to the different types of case to be included in each category. It must be clearly understood that this new conception proposed includes cases which could be found in any of the above-mentioned categories, but that it is not identical with any one of them.

It is proposed to call this new type of epilepsy "epileptic abreaction". Under this one intends to include those cases where the convulsive reaction is determined by faulty psychological reactions, for which it acts as an outlet. It must be emphasized most strongly that this is not in any sense a condition identical with that at present defined as "psychogenic epilepsy". This latter is a term used to signify cases, such as those described by Rows and Bond (1926), in which the initiation of epileptic reactions is in some situations powerfully toned with the emotion of fear, cases in which the epileptic manifestations are so essentially determined by fear that in some the very auræ derive their nature from the environmental conditions present at the time of the first attack, as in the case of the sailor whose aura consisted of an erythematous area on one cheek, corresponding to the impression of the pad placed against his cheek to take the recoil of the gun while occupied in circumstances which gave rise to his acute psychological conflict.

From the point of view of classification it seems unjustifiable to split off this so-called psychogenic epilepsy from other allied epilepsies in which psychological factors play such a determinant rcle. Pure psychogenic epilepsy, according to the present accepted definition of the term, is probably an extremely rare condition, though ultra-fashionable writing tends to deny this. The too whole-hearted statement in Rows and Bond's book that epilepsy is a functional nervous disorder cannot be regarded as anything other than a rather dramatic catchword stimulated by the current trend of psychology at the time the book was written. It seems a fallacious practice, at a time when the many epilepsies need some reasonably broad classification, to isolate, under a terminology far more precise and fixed than that applied to many other epilepsies of vastly wider incidence, an extremely small sub-group. One objects to this, not on the grounds that the definition is necessarily inaccurate, though the word "psychogenic" is rather conveniently ubiquitous, but that broad relationships within the multitude are possibly of more scientific

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use than the dogmatic labelling of a single sub-group, and a reliance on the blessed word "idiopathic" to consign the rest to the limbo of neglect.

Epilepsy and Hysteria.

It is usually carefully insisted that the convulsive reaction in psychogenic epilepsy is to be distinguished with the greatest circumspection from that in hysteria. The non-contamination of epileptic states with hysteria is insisted on not only for this particular variety of epilepsy, but in many other circumstances. It is an excellent rule, however, derived from the study of the psychology of the individual but applicable over the widest possible field, that where great care is being exercised to avoid what seem inevitable assumptions, it is wise to consider the possibility that these latter may be true. This helpful advice might well be followed in dealing with hysteria and epilepsy. It is almost impossible to take up a standard text-book in general medicine or neurology without finding some reference to the need for careful delineation between hysteria and epilepsy. It is extremely praiseworthy that, for so many decades, such extreme care should have been taken to dissociate conditions which obviously must present a surface similarity. It is, however, correspondingly mystifying to realize that, where so many similar manifestations exist in two conditions, the *fundamental* similarities in both cases have not been more closely insisted on. I raised this question at a scientific meeting, and was informed that the differentiation between the two was important because the treatment of the two conditions was different. There are two objections to such a standpoint. In the first case is it a sound scientific proposition to allow therapeutic considerations to preclude the formation of proper pathological conceptions? Does, for instance, the fact that the treatment of pulmonary fibrosis and acute lobar pneumonia is quite different, militate against the realization that in both there is an occlusion of vital lung tissue? In the second place, in certain types of epilepsy, i.e., in those cases which we will include under the heading of "epileptic abreaction", the treatment may not be so greatly different from that which is given to the hysteric.

There are several similarities between hysteria and epilepsy. Firstly we have the major convulsion itself, so similar in its broad outlines that decades of time, and oceans of ink, have been spent in proving their essential difference. Again, there are similarities to be discovered from a study of the personality of the afflicted individual. In both there is egoism and exhibitionist tendencies. Rows and Bond do well to emphasize certain temperamental properties characteristic of both types of personality. The presence, or otherwise, of an audience is by no means the infallible diagnostic adjunct that former writings would have us believe. Even in grossly developed institutional epileptics this exhibitionist tendency is well marked. Both epileptics and hysterics tend to have fits under emotional duress, particularly when some

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wish, or greatly desired course of action, is obstructed. In any large mental institution where an epileptic ward exists, the observer will always find a certain number of his patients in whom what we are prone to call unmistakably epileptiform convulsions are hysterical in their origin. This applies even more commonly among patients whom we would classify under the heading of "progressive epileptic deterioration". The initiation of convulsions in these cases is other than hysterical, but it may be that the early convulsions, by a process of facilitation, encourage a ready habit of discharge along pathways the stimulation of which leads to convulsions. This being so, other precipitants of fits are called into play, when the subject desires unconsciously to utilize the dramatic aspect of his disease to resolve those problems of which he is conscious. Hysterical symptoms, other than those of a convulsive nature, are present in what we call the indubitable epileptic; but more than this, the diseases are so intermingled that, while the distinction between a hysteric with obvious signs of his condition other than the epileptiform manifestations, and an epileptic, either markedly deteriorated or classifiable as a case of epileptic retardation, is not a matter of difficulty, more closely approximating aspects of epilepsy and hysteria can give rise to great diagnostic difficulty. The fact is shown by the diversity of nomenclature adopted in those aspects of each disease which border on one another, and which are presumed to be covered by the terms "hystero-epilepsy", "psychogenic epilepsy", etc.

In aspects of personality other than those directly elicited by ordinary personal observation, there is a definite similarity between the findings in hysteria and epilepsy. Those derived from the Rorschach test, which, in the author's opinion, is the most useful of all personality tests, bear this out to the full. It is impossible to convey, from considerations of space, any description of the technique of the Rorschach test, or of the similarity of its findings, but two very significant ones must be commented on. In the first place, both hysteria and epilepsy present most commonly what is called an unadapted extratensive *Erlebnistypus*. For a detailed description of the derivation and meaning of these findings the reader should consult Rorschach's *Psychodiagnostik*, and in English the works of P. E. Vernon (1933*a*, *b*, *c*; 1935) and Guirdham (1935). It may be said here that the *Erlebnistypus* signifies the reacting capacity of the individual. It does not convey the same meaning as is embodied in the terms "extraverted" and "introverted" according to, say, the definitions of Jung, as these latter represent fixed states already achieved by the individual, whereas Rorschach's *Erlebnistypus* conveys both innate possibilities of reaction as well as tendencies in regular operation. This is the baldest statement possible to make with regard to the *Erlebnistypus*. There is no word available in English which conveys the strict meaning of the German verb "*erleben*". For further information the above-mentioned sources of reference must be consulted about this, the most important of all the Rorschach conceptions. To say that a subject presents an unadapted

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extratensive *Erlebnistypus* means that the affectivity is objectively directed, that the subject is primarily extratensive, which conveys much of the meaning of the commoner description "extraverted", but that the affectivity lacks proper environmental adaptation, in virtue of the extreme egocentricity of the individual. This description applies both in the case of epilepsy and hysteria, both as regards the findings in the Rorschach test and those derived from personal observation. A further point of comparison, emphasized by the Rorschach test, but capable of derivation by other means, concerns the phenomenon of perseveration. Much work is being performed at present on this important but hitherto neglected psychological phenomenon. It is a condition remarkably demonstrated in the Rorschach test results of epileptics. I have no information as to its occurrence in the Rorschach results of hysterics, but information from other sources strongly suggests the possibility of perseveration being a very significant feature of hysterical conditions. Many hysterias are persisting echoes of the visceral accompaniments of fear, when the actual original stimulus evoking the fear has been withdrawn or forgotten. Not only this, but such hypotheses as the influence of the fixed idea, as in Charcot's theory, suggest that perseveration is an important factor in the genesis of hysteria. These facts are not offered because they are considered, of themselves, as in any way explanatory of the hysterical and the epileptic reaction. They are adduced only to emphasize the likeness between the two conditions.

Psychological Factors in Epilepsy in General.

It is as well to correct here any impression that epileptic abreaction is in any sense totally different from the other categories in being a psychological condition as distinct from a neurological one. Psychological factors play a part in practically all epilepsies. Psychological peculiarities are of the utmost importance in the genesis of one of the main categories, e.g., progressive epileptic deterioration. Apart from the above-mentioned psychological attributes acting as determining causes, psychological fluctuations in the environment are of great importance in all epilepsies, except those associated with such profound developmental disorders as are found with idiocy and the lower imbecile grades, and which are excluded from this classification.

The first psychological environmental influence to be dealt with is the effect of opposition. Practically all epileptics tend to have fits when their desires are in any way thwarted. This phenomenon is mostly evident in the higher types of epilepsy, meaning by higher those varieties accompanied by the least constriction of interest and the least epileptic deterioration. In less degenerated cases of progressive epileptic deterioration, and in more developed cases of essential epilepsy, the response is more crude and immediate, i.e., interference with the wishes of the patient leads to the immediate provocation

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of fits. In the case of epileptic abreaction the connection between the interference with the patient's wishes and the incidence of convulsions is not so close. Indeed, the very conflicts which are the basis of epileptic convulsions in certain cases of epileptic abreaction may require considerable investigation for their elucidation. This brings us to an important point—that so far as we are concerned with immediate psychological precipitants, as we pass from the lower to the higher of the four varieties of epilepsy we have mentioned, we find that these psychological stimulants of convulsive reaction are more obvious in the lower and less obvious in the higher varieties of epilepsy. We may recapitulate here that the order from lower to higher types is epileptic retardation, progressive epileptic deterioration, essential epilepsy and epileptic abreaction.

It must be clearly understood that the above refers only to psychological precipitants of convulsions. There is almost certainly some psychological *determining* cause in practically all the epilepsies. This is most doubtful in the case of epileptic retardation, but, even here, it probably applies. We have seen how there is a definite psychological determinant, in the epileptic characterological peculiarities, in the case of progressive epileptic deterioration. It is possible that it is only a question of time before this statement would apply to most epilepsies. (It seems hardly necessary to point out that this does not, in any way, obviate the undoubted potentiality of all the myriad biochemical and glandular etc. disturbances in their roles as excitants of convulsive reactions.) The important point to make in this connection is that, while all may be psychologically determined to some degree, in the case of progressive epileptic deterioration the reaction is on a lower level of consciousness than in the case of epileptic abreaction with its manifold hysterical complications. Epilepsy is a reaction on a lower teleological plane than that implied by the general meaning of the word "consciousness". The higher the form of epilepsy, the more phenomena associated with a higher degree of consciousness take part. Whatever the purists say, the cases of what were formerly called "hysterical epileptiform seizures" involved a far less deep loss of consciousness than in typical cases of epilepsy of the progressive deteriorative types. I feel sure that in a certain proportion of cases of the former type it is possible to inhibit the full play of the convulsion at different stages of its development. Here, then, is a second interesting fact—that the actual convulsive manifestations, in the higher epilepsies, are more closely approximating to the conscious level than the lower epilepsies.

EPILEPSY AS A LOWER TELEOLOGICAL RESPONSE.

It has been mentioned previously that determining psychological factors may exist in all the epilepsies. It must not be inferred from this that the author is of the school which insists on functional nervous disorder as a co-diagnosis with cerebral tumour, at that stage of development when the patient

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is going blind from the effects of the latter. One can safely use the word " psychological " in the lower epilepsies, without implying psychoneurosis in the accepted sense of the term, because here we are referring to psychological responses on a much lower plane. There is a great deal of evidence that epileptic reactions occur at the instinctive level, owing, among other things, to the epileptic's incapacity for the higher processes of abstractive synthesis. This limitation of much epileptic activity to the instinctive level is only part of a general regressive tendency displayed by epileptics. In other spheres there is a tendency to react on lower developmental planes. Thus religiosity has been explained (Goldblatt, 1928) as a return to the primitive religiousness of savages. There is little space here for the tabulation of the reasons for the above statements. The literature on the nature of the epileptic reaction deals extensively with such a question. But here it is necessary to interpolate my own opinion that, not only is epileptic behaviour as a whole redolent of the marks of reaction at a lower level, but also that the actual mechanism of the fit is essentially one hall-marked with the characteristics of instinctive behaviour.

The Nature of the Motor Phenomena.

According to the teleological view the almost involuntary and apparently purposeless writhings in pain witnessed in human beings are a relic of the reflex movements carried out by animals as a means of escaping from a noxious stimulus. MacCurdy (1925) has implied that all purposeless motility, which he calls restlessness, is an expression of anxiety. This perhaps would be better expressed as all surplus motility, in that it is almost certainly an overstatement to regard as purposeless that which reveals no present significance. (The general truth of MacCurdy's claim, it may be stated here, is questioned.)

It has been pointed out that the essential feature of the epileptic reaction is the withdrawal of interest from the environment, and Pierce Clarke (1912, 1926) has shown how this is revealed at its maximum in the unconsciousness coincident with the seizures. The connections of acute and specifically directed consciousness with motility have been freely discussed. MacCurdy (1925) has pointed out how the restlessness of anxiety states is a natural attempt to force a solution. The goal being withheld, the still operative conation results in an accumulated tension, as a result of which there occurs a meaningless motor discharge. He also suggests that in insomnia and epilepsy the movements present may be an attempt to maintain consciousness, but that their immediate effect, as in fear states, is to direct the patient's attention from his thoughts.

A more striking teleological analogy than the Darwinian theory of the connection between the writhing in pain of humans and the reflex movements

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of lower animals is offered by the convulsive syndrome in epilepsy. The two stages, the early rigidity and the subsequent clonicity, have all the simple, graphic, and self-limited characteristics of reflex action operating at the instinctive level. These motor adjustments and reactions are only purposeless when examined from the viewpoint which sees all manifestations of evident action as judged, not by the simple formulæ of instinctive requirements, but as adjusted in accordance with all the complexities of sentiment, judgment, etc., and, most effective of all, curbed and malformed by all the operations of repression. Regarded solely in their perceptual setting, the two motor stages of the epileptic phenomenon are purposive enough. In the tonic stage the rigidity represents graphically the immobility incidental to the first impact of fear. The instinct of self-preservation, where it exists unvitiated by the presence of other allied facts—perhaps only a theoretical possibility—is essentially accompanied, at the first operation of its related emotion of fear, by a muscular immobility which ensures an enforced attention to, and a greater knowledge of, the cause of the experienced emotion. The release of this tonicity in the classical clonic movements is a loosening of muscular tension and a mobility coincident with the operation of that second phase of the instinct of self-preservation which is known as flight.

Whether this explanation of the epileptic phenomenon as a compact self-limited and persisting primitive combination of the two closely allied fear mechanisms of rigidity and flight is, or is not, accepted, there can be no question that the motor phenomena of epilepsy show all the characteristics of reflex action. Such motor phenomena as the above are not incorporated in the specific nature and origin of an instinct. They are its pathway of discharge. What are the directing impulses behind such a reaction it is difficult to say exactly. Certainly fear must be the main determinant of this retreat to unconsciousness. For myself, I think that too little emphasis, in describing the separate stages of the fit, is placed upon the fact that an epileptic seizure is essentially a combination of the most profound and utter unconsciousness with the most evident and striking motor phenomena. (Here we are speaking of the epileptic discharge par excellence, as it occurs in progressive epileptic retardation.) In consideration of the teleological view of epilepsy, that it is a regression to an earlier developmental epoch, the view is tenable that the motor element in the fit is an exhibitionist phenomenon, dictated by the operation of the instinct of self-assertion. Such a view as this does explain the rationale of the motor phenomena. The retreat from fear into unconsciousness is an ubiquitous psychological mechanism. The expression and dispersion of fear in increased motility is also common. But why, when the subject has achieved his aim in becoming unconscious, does this very evident motor release occur? I suggest that, with the withdrawal from function of the higher centres, it is to be ascribed to the operation of the instinct of self-assertion at a lower reflex level. It might be argued that the presence of

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some self-conceiving instinct is necessary for the experiencing of fear, and therefore that we are describing as secondary a phenomenon derived from a mental unit which actually is primary. Such an argument, however, would be fallacious. Not only must what is called the instinct of self-preservation be, as McDougall has laid down, a later development, involving as it does the concept of self, than the instinct of flight,* but also the question involved in such an argument does not really arise. For while possibly, at our present evolutionary stage, the appreciation of fear demands, for its favourable operation, a clear conception of self, such a conception is, in primitive mankind, contained within the instinct of self-preservation, while in civilized communities it is probable that fear is most commonly aroused in connection with those feelings of moral self-censure incorporated in, and assisting in, the further development of the sentiment of self-regard. Again, while the instinct of self-assertion is a property widely diffused throughout mankind, its development in epileptics is to a degree so paramount that it is held, by those who have most carefully and scientifically approached the epileptic temperament, to be of all the most determining factor. It is probable, then, that the element in the fit which is derived from fear is the sudden cessation of consciousness, and that the subsequent motor discharge is due to the extinction from function of the higher centres, giving rise to a release phenomenon, expressive, in reflex activity, of the instinct of self-assertion. This tendency to react, from lower psychical levels, is general throughout the epileptic existence. Goldblatt (1928) asserts that it is demonstrated as a specific chronic tendency in the religiosity of the epileptic. There is abundant evidence, both from this test, and from observation, of the tendency to continue on the perceptual level, to react totally, to withhold nothing, to repeat previous reactive mechanisms in their entirety, and to derive from them no fundamental common denominator which might prevent these blind, repetitive actions, so characteristic of lower animal forms, yet so existent in the epileptic.

The two maximally operating forces would appear to be the instinct of self-preservation and that of self-assertion. The discrepancy of their separate operations constitutes the epileptic conflict. The aggressive impulse, derived from the instinct of self-assertion, is the one maximally operative in the early life of these subjects. Hence the recognition of the domineering, vain, boastful and aggressive epileptic as having existed as such before the onset of his fits. Later, with an increase in the element of fear, the instinct of self-assertion becomes more submerged. Its operations are hindered by the subconscious desire for extinction which is contained in the death symbolism of the epileptic convulsion, but its residual manifestations are revealed in the motor element of the fit itself. I cannot rid myself of the idea that there is a definite self-assertive and exhibitionist intention in the actual motor convulsion

* This is used in the sense in which it is employed by McDougall (1931), who postulates an instinct of fear as a more primitive possession than the instinct of self-preservation.

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of epilepsy. To regard the fit as a whole as an acme of the death wish, as a mechanism of escape dictated by fear, is logical enough. But why this classical specific motor discharge?

SELF-ASSERTION AND FEAR IN THE CONVULSIVE SYNDROME.

As to how far the movements incidental to the appreciation of anxiety, and those dictated by the instinct of self-assertion, play their part in the motor phenomena of epilepsy, it is impossible to deduce. We have argued that the clonic convulsions are expressive of the impulse of self-assertion operating at a low psychological level, on the plane of reflex activity. On the other hand, we have drawn attention to the striking teleological analogy between the two phases of the epileptic convulsion, the early rigidity and the ensuing clonicity, and the operations of the instinct of fear and its associated impulse to flight. My own standpoint is that these two views are not in any sense alternative and mutually exclusive hypotheses. It is my opinion that the impulse of fear may express itself in accordance with the degree of self-assertiveness present in the individual. Where the degree of self-assertiveness is strong, its tendency to muscular display is revealed even under the impulse of fear. It might be argued against this that all instincts must have a pragmatic value, that they are all dedicated to the conservation and continuance of the race, and that the manifest and demonstrative motility during the mechanism of flight would tend towards the obliterating of the subject manifesting it. There are innumerable reasons against such an objection. This excessive and self-revealing muscular display *does* occur in certain species under the operation of fear, as in the chattering and gesticulating smaller anthropoids, such as the chimpanzee. In the case of another instinct, at a higher level of development, an unbridled sex instinct tends towards social obliteration, but this does not preclude its manifestations in affected individuals. Again, the extinction of certain species appears to presuppose the non-productive nature of their instincts. Admittedly it is possible that here certain instincts reach a relative degree of development which does not obtain in the human species; but the common possession of the basic fundamentals of the principal instincts, however these are modified according to the level of evolution occupied by the different species, is a matter of which most psychologists are in agreement.

It is difficult to outline ætiological distinctions between epilepsy and hysteria. They are both components of a reaction-type in which are many often scarcely perceptible gradations. In both the hysterias and the epilepsies there is a prepotent presence of fear. The main definitive features in this group are as follows:

(*a*) In epilepsy and the motor hysterias, the instinct of self-assertion is displaying an opposing action. There is a dissociation between the desire

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for regression and the antagonistic action of the instinct of self-assertion. As we proceed to the more quiescent and paralytic hysteria types an accentuation of the submissive, in relation to the assertive instincts, is leading to the passive, retractive tendency, tending, in its full development, to hysterical paralyses.

(*b*) The combination of unconsciousness and motor discharge, as seen in epilepsy, occurs on an almost instinctive plane, and evolves its reaction largely on the perceptual level. The stimuli for its production are manifestly diverse, and almost certainly primitive, seeing how they are precipitated in subjects of such blunt perception that what must be considered emotional situations by higher types must often escape their notice.

On the other hand, the phenomena in hysteria are ideationally derived. They are initiated by repressed complexes; their overt motor manifestations are accompanied by a markedly demonstrated affectivity. They are centred round, and originally initiated by, some personal traumatic, either physical or psychical, experience. This latter is not the case in what we understand by idiopathic epilepsy. It obtains only in that very small and ever-to-be-scrutinized group of psychogenic epilepsies.

Psychology of the Epileptic State.

The importance of this psychological digression is as follows. The classification we have adopted is essentially a clinical one, but the elucidation of such problems as have been dealt with in the preceding paragraphs adduces further pathological evidence which might be available for the purposes of classification. What we have derived is the fact that reflex movements which, at the lowest teleological levels, accompany the activity of the instincts of fear and self-assertion, are at the root of the epileptic motor phenomenon. Fear is such an ubiquitous human possession that it may seem fallacious to make such specific mention of it, but the rigidity present in the first stage of the epileptic motor phenomenon is characteristic only of the epileptic reaction. Self-assertion, to the degree present in epileptics, is abnormal, and characteristic of this disease, even if we are only considering the undramatic, non-conclusive attributes of personality. The display of clonicity, as representative of self-assertion at the reflex level, is quite characteristic.

The above peculiarities supply evidence applicable to the epileptic reaction as a whole, independent of the particular clinical variety it assumes. These reactions are manifestations of fear and self-assertion. In differentiating the one from the other such a generalized property as fear is of little use. As regards self-assertion, it would be fascinating if we could postulate degrees of this property to account for the different clinical categories. This is quite out of the question. We have not at our disposal the means of assessing the relative strength of the primary instincts; but it is possible to distinguish

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between the different categories we have defined according to the degree to which the psychological determinants are of importance in giving rise to the fits, and according to the degree of adaptation presented.

In the case of epileptic retardation the psychological peculiarities are less marked than in the other categories, and probably play a smaller part in precipitating the convulsions. In the case of progressive epileptic deterioration the temperamental peculiarities, chiefly with regard to the abnormal development of the self-assertive instinct, are seen in their purest culture. The constriction of the sphere of interest shown in these cases limits, in a typical case, the expression of the subject's fundamental deficiencies to the typical epileptic major convulsion. In the case of essential epilepsy the intellectual and reactive personalities of the individual are so variable that it is impossible to be dogmatic. There are cases where the characterological peculiarities border on those displayed by the progressively deteriorating epileptic. Abreaction, on the other hand, is a condition where the dominant self-assertiveness does not always express itself on the low teleological plane which is customary in progressive epileptic deterioration. When it does so the patient is often more accessible than in the latter condition, so that stages of the attack may be inhibited. On other occasions the seizure may be replaced or contaminated by epileptiform hysterical symptoms. In these there may be a pseudo-purposeful display of emotions other than those accompanying the epileptiform reaction. Though the emotional varieties of hysteria cannot be said to occur at any very high developmental level, they at least enjoy a co-operative emotional display more complex, and more tending to a practical solution of the patient's difficulties, than in the case of progressive epileptic deterioration, in that in hysteria we are dealing with a condition which more purposely advertises itself to its environment, and which makes closer and more subtle contact with available sources of sympathy.

The Alliance of Physical and Psychological Causes in Epilepsy.

In closing this section it must again be emphasized that, in discussing the psychological accompaniments of epilepsy and their possible function as determining factors, one is not losing sight of the function of the well-authenticated physical stimuli as precipitating, or even as determining, factors. The point one considers of greatest importance to make here is that the physical factors in epileptic causation, while undoubtedly proved to apply in individual cases, and, moreover, in different types of case, are nowhere absolutely specific for epilepsy. Alkalosis occurs in epilepsy, and correction of this condition will ameliorate some epilepsies; but alkalosis also occurs elsewhere. Protein sensitivity occurs in epilepsy, but it occurs in asthma, migraine, etc. Indeed the diversity of the epilepsies prevents us from thinking that any single

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collection of pathological* abnormalities will ever be found to account for the signs of epilepsy in all the forms in which it occurs. But the above arguments do serve to show that the assumption that epilepsy can be fully explained by postulating first an inherent irritability of the central nervous system, and secondly some physical stimulating factor, is inadequate. The coincidence of some psychological factors is too strong to be neglected. That it provides an interim between an inherent convulsive tendency and a series of physical stimuli is too ready a solution of a difficult problem. In the present state of knowledge it is better to admit that the interrelations of physical and psychological factors are not thoroughly elucidated, but that both are operative.

Diseases Related to Epilepsy.

It may be asked why no mention has been made of certain other symptom-complexes allied to, and sometimes alternating with, epilepsy, e.g., migraine. The reason is twofold. Firstly, in migraine much of the reason for classifying the two diseases together is that both are assumed to rest on a common basis, i.e., on allergic factors. This viewpoint must apply only to a limited number of cases of epilepsy. The second reason, applying both in this case we have specifically mentioned, and in others which might be adduced, is that we are in this thesis investigating, not the pathological relationships of any particular brand of epilepsy, but the classification of such conditions where an innate tendency to convulsion is manifested. For this reason such manifestations as migraine, together with so many conditions referred to under the heading of "epileptoid" by E. Bleuler, and as affect epilepsies by several of the German authors, are here excluded from notice. The states referred to are epileptic fugues, the various psychological equivalents, etc. Epileptic they may be. Convulsive they are not. Besides, with the exception of certain epileptic furors, often found in association with the fits, usually succeeding them as in post-epileptic confusion, these psychological states allied to epilepsy are, in their psychological relationships, associated chiefly with the epileptic abreactions, and possibly some varieties of essential epilepsy. I consider that such fugues, etc., should only be included in this classification when epileptic convulsions are also present.

Conclusion.

Most classifications of epilepsy are merely categories of exciting factors, or labels emphasizing an inadequately elucidated pathology. Present systems of classification emphasize too much a minority of epilepsies in which psychological factors play a part. These latter are more uniformly diffused through epilepsy as a whole, it being insisted that this condition is not a disease, but a

* By this one means pathological in the sense of physical disease.

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peculiarity of response on a lower developmental plane. Four clinically distinguishable varieties of epilepsy are described.

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THE TREATMENT OF EPILEPSY WITH PSYCHOSIS BY PROMINAL.

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PROMINAL was first favourably reported on by several German and other continental observers (1, 2, 3, 4) in 1932. More recently (6, 7, 8, 9, 10) there has been evidence of its value from workers in this country, mainly of its use in epileptics with mental defect. In the reported cases results have indicated that only 3 out of 66 cases failed to show diminution in the number of their fits. In the epileptic psychotics treated at Claybury during the past eighteen months the drug has been found useful in a less striking way.

All the cases shown here had already received treatment for long and varying periods with either phenobarbitone or bromides or a combination of these drugs. Owing to the practical consideration that withdrawal of treatment for control observation would not have been justifiable, the results only provide a running comparison between the efficiency of prominal, phenobarbitone and bromide. The last-named two drugs have been used separately in 15 cases and combined in 11. The features of an epileptic psychosis were exhibited in every case, with frequent fits, mainly of the grand mal type. The fits began in infancy in 5 cases, at puberty in 15, in adult life in 4, and in 2 the time of onset was unknown. Eighteen cases were capable of various occupations.

The close chemical alliance of prominal with phenobarbitone is shown by its formula, sodium methyl-ethyl-phenyl-malonylurea. The change from phenobarbitone and bromides to prominal was effected gradually over a period of 12 days. One-third and later two-thirds of the phenobarbitone or phenobarbitone-bromide dosage was replaced by prominal before the transition was completed. Polstorff's estimation that 3 gr. of prominal (1 tablet) are equivalent to 15 gr. of potassium bromide or 1½ gr. of phenobarbitone was taken as a basis in assessing the dosage in each case. Alterations in dosage were eventually found necessary to suit individual cases. In these patients relatively high doses were required in order to achieve the best results. Six grains twice daily were well tolerated in the cases with most frequent fits, while 3 gr. sufficed for the milder ones. This dosage did not cause drowsiness or loss of working ability such as would have occurred with a corresponding dose of phenobarbitone or bromide.

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Case No.	Treatment period of each drug in weeks.	Phenobarbitone. Fits. G.M.	P.M.	Total.	Prominal. Fits. G.M.	P.M.	Total.	Actual increase (+) or decrease (−) in number of fits.	Percentage increase (+) or decrease (−) in number of fits.	Physical health.	Behaviour.	Working ability.
1	52	43	39	82	21	52	73	−9	−11	+	+	+
2	52	34	0	34	6	0	6	−28	−83	+	+	0
3	21	12	22	34	17	20	37	+3	+9	0	0	0
4	21	18	0	18	12	0	12	−6	−33	0	0	0
5	39	14	15	29	8	10	18	−11	−38	0	0	0
6	19	41	0	41	44	0	44	+3	+7	0	0	0
7	39	78	0	78	14	0	14	−64	−82	+	+	+
8	42	71	1	72	149	0	149	+77	+110	−	−	0
9	78	19	2	21	11	0	11	−10	−49	0	0	0
10	10	95	0	95	26	0	26	−69	−70	0	0	0
11	16	17	0	17	31	15	46	+29	+170	0	0	0
		Phenobarbitone and bromide.										
12	52	238	8	246	225	3	228	−18	−7	0	+	0
13	8	74	0	74	20	0	20	−54	−73	+	+	+
14	52	146	33	179	49	2	51	−128	−71	+	0	0
15	52	128	9	137	91	6	97	−40	−29	+	+	+
16	42	90	0	90	62	0	62	−28	−31	0	+	+
17	16	16	0	16	20	0	20	+4	+25	0	0	0
18	17	17	11	28	24	13	37	+9	+32	+	0	0
19	26	68	2	70	40	3	43	−27	−38	−	−	−
20	21	104	22	126	109	1	110	−16	−13	0	0	0
21	21	105	27	132	176	5	181	+49	+37	−	−	0
22	21	23	25	48	30	18	48	0	0	0	0	0
		Bromide.										
23	21	14	1	15	14	2	16	+1	+6½	+	0	0
24	21	16	4	20	16	20	36	+16	+80	0	+	+
25	18	36	0	36	26	0	26	−10	−28	0	−	0
26	21	31	0	31	48	0	48	+17	+55	0	−	0

Improved = +; no change = 0; worse = −.

Results on a Percentage Basis.

No change indicates an alteration of less than 10%.

Drug.	Fits. Improved.	No change.	Worse.	Number of cases.
Phenobarbitone . . .	7	2	2	11
Phenobarbitone and bromide	6	2	3	11
Bromide	1	1	2	4

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In comparing results the following points were observed:

1. Fits—number and type, etc.
2. Behaviour.
3. Physical health.
4. Capacity for work.
5. Toxic effects.
6. Duration of effect.
7. Leucocyte variations.

Commenting on these features, it is noted that in no case did the fits cease entirely during the use of prominal, though Jekelius (5) has reported this to occur in 40% of cases. In two cases, Nos. 3 and 16, the fits have swung from the major to the minor type. One of these, No. 16, has lost much of the stupor and mental confusion which were formerly an unusually marked after-feature of her fits. Petit mal attacks are less influenced than grand mal by prominal.

Physical health tended to improve where the fits decreased. Similar observations were found with regard to ability in following occupations.

Attempts to compare the efficacy of bromide, phenobarbitone and prominal by the use of intelligence tests proved impracticable.

Toxic effects were very infrequent. Drowsiness occurred in a very few cases shortly after the beginning of treatment, but rapidly passed off. One case complained of a dry mouth in the mornings. Another case has shown weakness of the lower limbs and impairment in walking since the use of prominal. Her mental state has deteriorated and the fits have increased, making her true walking capacity difficult to assess.

The duration of effect was at least as long as with phenobarbitone or bromides.

In one case (No. 22) the onset of regular menstruation coincided with the exhibition of prominal. Menstruation was formerly irregular and infrequent in this case.

Blood examination, consisting of white cell counts, both total and differential, were performed at the start and finish of prominal treatment in 80% of cases. The examinations were performed between 8 and 9 a.m., the patients being in a fasting condition, and as far as possible removed from disturbing influences before being tested. Ten cases showed a rise in total white cell counts of over 25%. Nine of these had previously been receiving phenobarbitone. Three cases formerly on bromide alone showed no appreciable change in white cell count. No significant alterations were noted in the differential counts.

In estimating the value of prominal, the natural variability of epilepsy should be taken into account. It is said that this disorder improves in the summer. In the majority of these cases this factor favours the control period. Some epileptics tend to improve for a time on any new medicine,

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nevertheless it was found that those cases in which the fits diminished during the first four weeks of treatment were likely to maintain their improvement over longer periods.

In cost, prominal is equivalent to the proprietary luminal, but is considerably more expensive than phenobarbitone B.P.

Prominal does not prevent the progress of mental deterioration where this is already in evidence. A decrease in number of fits is not necessarily followed by an improved mental state. Suppression of fits in two cases appeared to make their mental condition worse. In patients whose mental disorder was of a less severe nature prominal gave better results, both as regards fits and behaviour, while in those already subject to severe mental disturbance the drug was less effective than phenobarbitone or bromide in diminishing fits and retarding the decline of their mental health.

Neither the number of years during which a patient had suffered from epilepsy nor the actual frequency of fits gave any indication as to the possible benefit to be expected from prominal. A trial of at least two months appears to be the only way of finding out its effect with certainty in a given case.

Conclusions.—These examples of idiopathic epilepsy in psychotic patients showed—

1. That prominal was active in a majority in reducing the number of fits.

2. That improvement may be obtained mentally, physically and in occupational capacity in patients showing less severe degrees of mental disorder.

3. That the more advanced insane epileptics are unlikely to be benefited, and may be made worse by its use. Phenobarbitone or bromides are the more useful with this type.

4. Confirmation of the idiosyncrasy of epileptics to different drugs, and the necessity for dosage on a strictly individual basis.

I wish to express my thanks to Dr. G. F. Barham for his help and advice, and for permission to publish this work.

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FAMILIAL PSYCHOSES ASSOCIATED WITH ENDOCRINE DISORDER.

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ONE of the most striking characteristics of patients in a mental hospital diagnosed as suffering from schizophrenia, or dementia præcox, is their dissimilarity. It is easy to pick out cases in which the onset, course, symptomatology and outcome of the psychosis are different. There is thus a tendency to regard the schizophrenic group as possibly containing many different clinical entities. One of the current major problems of psychiatry is to clarify what constitutes schizophrenia and to subdivide the clinical material further in a way which will prove useful for study.

The following two case-records of a sister and brother suffering from schizophrenic-like psychoses exemplify these points. The woman, who had been diagnosed as a case of dementia præcox, was examined in the course of a survey of many patients in a large mental hospital. She suffers from an atypical psychosis associated with endocrine disorder. Her brother has a similar psychosis, but lives out of hospital. The parents were first cousins. I suggest that these two patients suffer from the same disease process, that the course and symptomatology are definite enough for the syndrome to be considered a clinical entity, and that the syndrome is in part determined by a recessive genetical factor.

A. B—, a female, now aged 60, was sent to hospital at the age of 49, because her behaviour was so disordered that she was unable to take care of herself.

The history of the illness was that, about the age of 40, hair began to grow on her face, and she became obese. Five years later, in response to an urgent letter, a relative visited the family and found the patient "very insane". She was "fat, lazy, stupid, irresponsible", and had not worked for some years. She and her brother were being cared for with much difficulty by their aged, widowed mother. Four years later the mother died. A relative found the house in a state of dirt and disorder. A. B— had been trying to force food down her mother's throat. She was sent to hospital that evening.

The history of her early life is scanty, but reliable as far as it goes. She was a healthy girl, of average intelligence, reached Standard 6 in school by age of 14, and later earned a living as a dressmaker. She is said to have been of a cheerful disposition. She took much interest in helping with "school-treats" for children. She did not marry. A photograph at the age of 35

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shows her to be a good-looking, well-proportioned, though short, woman, without any excess of hair or fat.

On admission, aged 49, she was 5 ft. 1 in. high, and weighed 11 st. 4 lb. Her body was dirty and neglected. She had a profuse beard, at least 6 in. long. She was very stout, and some years later she was operated on for a right femoral hernia. Menstruation had ceased before admission, and her menstrual history is unknown. The mental picture was one of gross inactivity, indifference, personal neglect, and preoccupation with a vague, unsystematized delusional system, of erotic trend.

She realized she was in hospital, and recognized doctors and nurses as such; yet she was "incapable of giving any account of herself". Her answers were often obscene and irrelevant, and she muttered much to herself. She was unemployable, defective in personal habits, hoarded rubbish in her clothes, and sat about, often "tailor-wise", in happy, preoccupied indifference. A "speech-defect" was noted.

She said she had one sweetheart and one husband and one wife, and that was all she wanted. She said she was 17 years old, and was related to royalty. She was older in the sun than in the shade. Some years later she maintained she was "born this morning", was married and had millions of children.

Her condition has changed very little in the eleven years since admission.

EXAMINATION.

Mental: Behaviour.

At the age of 60 she presents the same picture of inactivity and indifference. She can dress herself, but has to be washed. Her personal habits are frequently defective. On occasion she can do plain sewing quite well, but usually she sits muttering to herself or wanders about picking up rubbish. She recently hid a dead rat in her clothes. She rarely talks spontaneously to other patients, and she never looks at newspapers. She likes the radio music. She is quite observant of her surroundings. She is not nearly as inaccessible as her greatly disordered appearance might suggest. She at once obeys a request to come for examination, she understands questions well and can answer briskly and to the point. Frequently, however, she lapses into a low muttering on her delusional topics, from which it is hard to distract her. She often twiddles the fingers of both hands in front of her face, and is unable to give a coherent explanation of why she does this.

> Physician looks at her intently. Patient: Do you recognize me, sir? [laughs to herself].
>
> Dr.: What's your name? [Correct answer.]
>
> How old are you? Born this morning.
>
> What do you complain of? —— Nothing [laughs]; then, "My sweetheart" and other mutterings.

Affect and Content.

She is obviously happy. She sits smiling, enjoying the interview. In the ward she is usually content, sometimes elated or indifferent, but never obviously sad. She is irritable if crossed.

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How are your spirits? —— Dull now and again.

How do you feel? —— I don't feel as if I'd like to go away from here. I think it's a nice place.

Are you happy? —— Yes.

Why? —— Perhaps because you're here [laughs]. I've been a gentleman in the army. I've been Lord Kitchener.

Are you a man or a woman? —— I'm a lady now. I've been a gentleman.

But why are you so happy? —— Because I am. Because I have a lovely husband and a true one.

She calls her husband "Mr. Algy", but can give no more facts about him. There is no evidence of hallucinatory visits from him.

Do any funny things happen to you? —— You mean about the Devil?

Yes? —— The Devil put his arm round my waist, and he said, Go in that way. He said Ha! Ha! Ha!

Do you ever feel as if people were speaking to you? —— Liberals out in the street voting.

Have you ever heard them? —— I have a lot of children, pretty girls.

She sometimes refers to herself as Queen Annie of Hyde Park and Piccadilly Circus. She cannot be exact about her delusions and hallucinations. They are vague and not formulated into any coherent system.

Sensorium.

Orientation.—She knows she is in hospital, but calls the hospital by her own name. She thinks she is in Birmingham [incorrect]. She gives the date as "28 March, 1881—no, 1889" [correct date is May 8]. There is thus much disorientation to time and place.

Memory: Remote.

How is your memory? —— Getting better. I lost my memory; I found it one day.

Where were you born? —— London [correct].

Patient then asked how old the doctor was and where he worked. She believes her parents are still alive and living in her old home.

When were you born? —— This morning.

Birthday? —— 25th October.

Year? —— 1881—no, 1884.

Day to-day? —— 28th March.

Where to school? —— G— Street [correct]. Oxford and Cambridge [incorrect].

How old on leaving? —— 14 [correct].

What work after leaving? —— Dressmaking [correct].

She can give some slight account of her work.

Did you like that? —— Yes. I got nervous debility. I've had nervous debility.

How does it affect you? —— Shaky legs. [She cannot explain this further.]

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Recent Memory.

When did you come here? —— This morning—when we had our hair long.

How long been here? —— I don't know; since the day they asked me to come; about three years [eleven years].

What has been done for you to-day? —— Nothing—my face—a clean frock.

Name of charge nurse? —— [Correct].

Her memory is diffusely impaired, especially for events of more recent years since the onset of her psychosis.

Power of retention.—She can remember "a pencil and a green tie" after five minutes.

Intellectual functions.—She can repeat only 5 figures forwards and 3 backwards; yet she can perform Marie's Three Paper Test at once correctly.

She can count from 1 to 20, and from 20 down to 1, can repeat the ABC and the months of the year correctly. She gives the King's name as "Mr. Algy", the Prime Minister as Lord Salisbury, five large towns in Britain as "London, Islington, Great Euston Road, St. Pancras", and five rivers as "River Swee [correct], River Wye [correct], River Dover [incorrect]".

Asked to substract 7 from 100, she begins "93, 87, 82", and stops. Her reading age on Burt's reading test is 11½ years, with a big scatter from the eight-to thirteen-year levels. She reads a simple story slowly and correctly, but is able to repeat very little. She writes her name easily.

She cannot explain simple differences, such as tree and bush, wood and glass, etc.

Insight.

Any need for you to be in a mental hospital? —— No, not after you have done all these things.

Are you ill in any way? —— Well, I have a pain in my leg.

There is some intellectual defect, but owing to some lack of co-operation and delusional preoccupation the defect may not be as great as the results in the test suggest. The scatter in the reading test suggests deterioration. She has no insight into her present state, yet she knows she has had an illness; "lost my memory, nervous debility".

Physical.

Her habitus is abnormal (see Figs. 1 and 2). She is broad and short, with a large round head and small hands and feet. Her height is 5 ft. 1 in., and she weighs 11 st. 7 lb. The shoulders are broad, yet the pelvis is wide and the thighs incline inwards at a feminine angle. The legs are short. Her span is 4 ft. 7½ in. The limb muscles are small and flabby. Fat is excessive everywhere. It is deposited most profusely on the hips, abdomen and chest. The arms and legs, fingers and toes taper towards the periphery. She has a well-marked beard and moustache of coarse, thick, dark hair turning grey. Her head hair is profuse, but coarse and thick. Her beard is cut once a week. Axillary hair is profuse. Pubic hair is well developed and of feminine distribution. There is much hair down the inner side of the thighs and legs. The body-hair is not over-developed. The breasts and labia majora are well developed.

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The clitoris is not enlarged, the uterus is not palpable. The skin is very dry. The dorsum of the forearms and hands acquires a marked sunburn in summer. This brown pigmentation is darker than her facial sunburn. It is absent in winter. She has had no dermatitis.

The ears are large, but well formed. The palate has a very high and narrow arch. She has about twenty small brown pigment-spots like dark freckles on the face, and four small pedunculated epidermal nodules on the right eyelids.

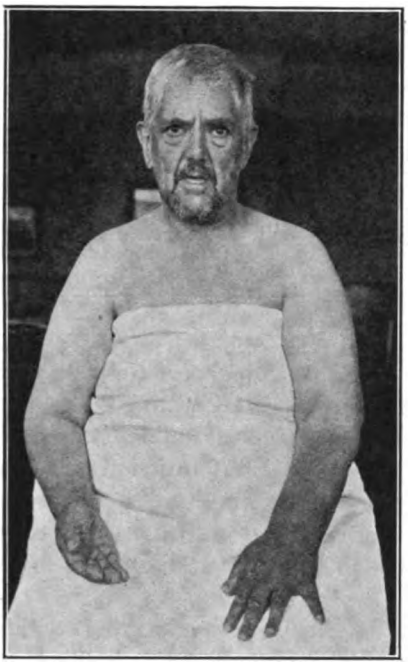

FIG. 1.

Female patient, A. B—, aged 60.

FIG. 2.

The heart-beat is regular in time and force, 82 per minute; the blood-pressure 252 systolic, 114 diastolic. There is no thickening of the radial or retinal arteries. Owing to the obesity it is hard to tell the size of the heart; it is certainly not grossly enlarged. There is a soft aortic systolic murmur, with an accentuation of the aortic second sound. The urine is acid, of specific gravity 1020, and contains a small quantity of albumen, but no sugar and no acetone. By microscope some leucocytes, epithelial cells and a few red blood-cells can be seen. There are no casts.

The abdomen is lax and protuberant. Striæ are prominent. No tumour can be felt. The liver and spleen are not enlarged, the kidneys are not palpable. The lungs are healthy.

No disorder can be made out in the nervous system. Her gait is slow and shuffling, but steady. She has a slight kyphosis. Her articulation is slurred,

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but she is edentulous. The cranial nerves are normal. In particular there is no disorder of smell, the fields of vision are full, the retinæ healthy. Visual acuity is slightly reduced in both eyes, but is difficult to examine. The pupils are equal, circular, slightly irregular, but react well to light and on accommodation. The ocular movements are free, without nystagmus. There is no muscular weakness, inco-ordination or tremor.

The blood Wassermann is negative, blood urea 25 mgrm. per 100 c.c., blood cholesterol 298 mgrm. per 100 c.c. serum.

The glucose tolerance test is within normal limits:

Fasting blood-sugar level before breakfast	100 mgrm.	sugar per	100 c.c.
Blood taken 45 min. after feed of 100 grm. glucose . .	136 ,,	,,	100 ,,
,, ,, 1 hr. 15 min. after feed of 100 grm. glucose .	122 ,,	,,	100 ,,
,, ,, 1 hr. 54 min. ,, ,, 100 ,, ,, .	115 ,,	,,	100 ,,
,, ,, 2 hr. 16 min. ,, ,, 100 ,, ,, .	96 ,,	,,	100 ,,

The blood-count showed: Red blood-cells, 6,120,000 per c.mm.; white cells, 6,200; hæmoglobin, 110%; colour index, 0·9. The differential count showed: Polymorphonuclear leucocytes, 69%; small lymphocytes, 26%; large lymphocytes, 4%; hyaline cells, 1%. A radiogram of her skull showed no abnormality of the pituitary region.

The case may be summarized thus: A healthy woman of cheerful, active disposition, at about age of 40, shows an insidious onset for no obvious cause, of hirsutism, obesity, increasing inactivity and emotional indifference, leading to a psychosis characterized at age of 60 by a gross deterioration of behaviour, personal neglect, emotional blunting, vague, poorly formulated, grandiose and erotic delusions, some intellectual decay, diffuse defect of memory, disorientation, and lack of insight into her present state, but a realization that she has had a "nervous debility".

Her physical state is characterized by a markedly broad and short habitus, a large head and small hands and feet, slight kyphosis and shuffling gait, obesity, hirsutism, marked hyperpiesia not of cardiac or renal origin, undue pigmentation of forearms and hands, an increase of blood cholesterol and of red blood-cells.

The brother, B. B—, presents a similar picture:

Only a scanty history is available. Born in London, a year before his sister, he reached the sixth standard at school and then worked in an umbrella factory for about eight years. Apparently he was a healthy boy. A deterioration of behaviour set in about the age of 23, since when he has never been out of the care of his relatives. A relative who saw him once in his later twenties found him at that time "a bit simple and soft". Boys used to tease him. His speech was slurred and he had a shake of the arms. He was not working and was cared for at home. In later years, apparently, his behaviour was worse than that of his sister, as his mother used to keep him locked in a room.

Since the age of 50 he has lived on a small farm and been cared for by a

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cousin. She has noticed little change in his condition over the past eleven years. If anything, his behaviour has improved slightly under her strict discipline. Whereas formerly he used to roam about and smash windows, now he is more orderly, obedient, and does a little work.

Examination.

Mental : Behaviour.

There is, however, at the age of 61, a gross disorder of his behaviour. The limit of his work ability is to pump the well and clean out the stable. Over these jobs he will spend hours. Unless supervised, he will stand about doing

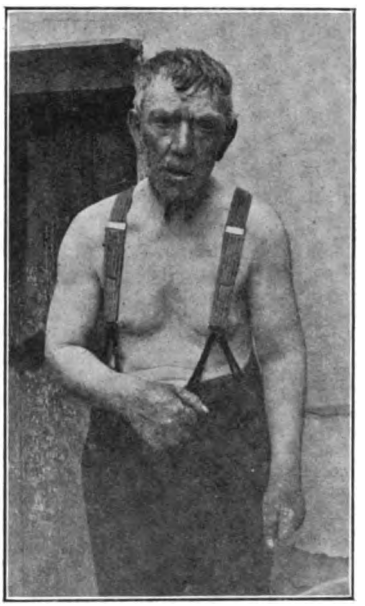

Fig. 3.—Brother, B. B—, aged 61.

nothing or muttering to himself, like his sister. He frequently puts his clothes on inside out. He rarely washes himself. Like his sister, he will defæcate and urinate anywhere. He used to urinate in his tea and drink it. He requires an almost constant supervision. He has a huge appetite. He can use a spoon and fork, but spills his food. He picks himself. He occasionally reads newspapers, and will comment on the horse-racing news. He can play a simple card game, which consists of pairing cards of like number. He recognizes the tunes of a few gramophone records. He often forgets what he is told to do. Usually dull, placid and contented, he will occasionally display a little querulous irritability. He is afraid of going out in the dark.

He recognizes the doctor as such, and he answers questions slowly and after delay.

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Affect.

He says he is "all right" and "quite happy". There is no elation. He is dull, indifferent and contented. There is no evidence of delusion or hallucination.

Sensorium.

Orientation.—He knows the month and the year, but not the day or date. He knows what county he lives in, but not the name of the farm.

Memory.—He thinks his memory is "all right". He tells his full name correctly, but gives his age as 63, and does not know his birthday or birth year. He names the street in London where he was born, and the school he attended. He says he left school at age 16 (?), from the sixth standard. He can give only a slight description of his work in the umbrella factory ("a good trade, better than farming"), but he says he left because he "could not get work".

His memory for recent events shows similar defects. He can describe his work of to-day, but not of yesterday. He can remember the physician's name for three minutes, but not an address.

Intellectual functions.—He can repeat six figures forwards and four backwards. He does Marie's Three Paper Test on the second attempt. He can repeat the months of the year and the Lord's Prayer. He knows the name of the Prime Minister, and the names of five large towns, but of only one river. He can count from 1 to 20 and from 20 to 1. He subtracts 7 from 100 serially in 1 min. 45 sec., with six mistakes. He can write to dictation. His reading age on Burt's Reading Test is 13·2 years, with a scatter from 12 to 14. He can read a short story, and repeat it correctly. He can describe the difference between wood and glass, a mistake and a lie, but not between poverty and misery. He performs all these tests slowly and with effort. He has very little knowledge of current events.

Insight.

He does not appear to be aware of the abnormality of his behaviour, and he says he does not feel different from other people. He does not consider his defective personal habits unusual.

Physical.

There is a facial and bodily resemblance to his sister, but he is less broad and short. His height is 5 ft. 4½ in. His gait is clumsy, unsteady, irregular. He walks with a stoop, and sways a little.

His hands and feet are not small relatively to his limbs. His head is large. The ears are large and long. The palate has a high arch. The irides have a colouring very similar to those of his sister. The head hair is dark brown, coarse, plentiful. He has a fair, reddish moustache. The body hair is not excessive, and has a masculine distribution. The skin is very dry. Fat, though plentiful everywhere, is not excessive except in the breasts. The penis is small and short. The testicles are normally developed. The hands are darkly pigmented in summer, and show a severe dermatitis, which appears in summer and dies away in winter. The skin, both on the palms and on the back of the hands, is much thickened and fissured. In many places, especially on the back of the fingers and thumbs, are ulcers exuding serum and blood, and having thick, raised whitish edges.

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The blood-pressure is 168 systolic and 88 diastolic. The heart is not enlarged, there are no cardiac murmurs, and no sclerosis of the radial or retinal arteries. The urine, S.G. 1030, contains 0·1% albumen, but no sugar or acetone. Blood and renal epithelial cells can be seen by microscope. There are no abdominal tumours. The liver is not enlarged. The kidneys are just palpable; they are not tender. For many years evidence of seminal emissions or of masturbation has been lacking. The nervous system shows little abnormality. His articulation is slightly thick and slurred. There is a slight coarse tremor of the protruded tongue and of the outstretched hands. The gait has been noted. There is no Rombergism, no inco-ordination, no loss of sensation, no muscle weakness. The tendon reflexes are active and equal, the abdominal reflexes present and equal, the plantar responses flexor. The cranial nerves are normal. In particular, the fields of vision are full, the vision normal, and the retinæ healthy. The pupils are round, regular, equal and react to light.

Neither patient has suffered from headaches, diplopia, somnolence or disturbance of urinary output.

To summarize: A healthy boy, of average intelligence, from age 23 onwards became increasingly inactive and indifferent, so that he was unable to work and had to be looked after at home. He developed slurred speech and a tremor, and his behaviour steadily deteriorated. At age 61 he is the victim of a psychosis characterized by a gross deterioration of behaviour, personal neglect, lack of initiative, emotional blunting, diffuse defects of memory, disorientation, a slight intellectual deterioration and a marked lack of insight.

Physically he is of a broad and short habitus, with a large head. He has a slight kyphosis, an unsteady gait, coarse tremor of the hands and tongue, and slurred speech. He also shows a moderate obesity, a lack of sexual activity, hyperpiesia and an undue skin pigmentation of the hands, associated every summer with a severe dermatitis.

The Family History.

Sibs of patient: One child died about 3 months old, cause unknown.

Parents:—Father: A horse slaughterer; a large, tall man, always healthy. Died, æt. 68, from "dropsy". He is described as "peculiar" in temperament. Although efficient at his work, he was very bad-tempered and unfriendly. In his later years he sat about the house doing nothing; often silent for hours.

Mother: A very small and short, hard-working woman, with a cheerful and stable personality. Died aged 86.

The parents were first cousins. The father's mother was sister to the mother's mother.

Paternal grandfather: Unknown.

Paternal grandmother: Died old; said to have been healthy.

Maternal grandfather: Normal; died 63.

Maternal grandmother: Normal; died 74. Sister of paternal grandmother.

Great uncles and aunts unknown.

Paternal uncles and aunts: Unknown. One uncle is said to have been "simple-minded".

Paternal cousins: Unknown.

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Maternal uncles and aunts and first cousins:

(1) Uncle, healthy, died over 60. Several children, unknown.

(2) Uncle, healthy, died 74. Eleven children; 5 died in infancy; 3 normal males died, aged 36, tuberculosis; aged 46, tuberculosis; and aged 65, cancer. Two normal males alive, ages 45 and 48. One female, aged 58, married.

This female cousin was examined. Always a worrier, with obsessional trend; worse since menopause at 45. Has always worried about religion and about rightness or wrongness of her actions. Attended a London hospital intermittently for four years for treatment as out-patient. Is now preoccupied with question of whether she should be baptized, but wonders whether, if she were baptized, she would stop worrying or worry about something else. When worried, she often has "an awful pain and pressure on the top of the head, queer feelings, and tingling in the arms". She has always been able to manage her house for her husband and children.

(3) Uncle, alive, aged 78. Enlarged prostate removed at age of 71. Now mildly senile, with poor memory.

(4) Aunt, healthy, died 75. Three male and 2 female children alive and well, over age of 50. One male died from accident in the thirties.

(5) Aunt, healthy, died "middle life". Three children: boy and girl adult, alive and well; one girl died in childhood.

(6) Aunt, died over 70 years. In her later years she became much preoccupied with religion, shut herself in her house and refused to see anyone. This is said to have followed her finding that her husband was unfaithful. Children: Three boys and one girl, alive and well, all adult.

COMMENTARY.

These two patients have similar psychoses. The onset is insidious. They both show a progressive deterioration of behaviour and an increasing emotional blunting, to such an extent that they cannot take care of themselves. Both show some intellectual decay, defect of memory, disorientation and lack of insight.

Both show a similar abnormal physique, a kyphosis and a shuffling gait. Tremor and slurred speech is observed in the man, and a speech defect was at one time noted in the woman. Both show other abnormalities strongly suggestive of an endocrine malfunction: obesity, hyperpiesia, skin pigmentation, diminished sexual activity in the man, and hirsutism, high blood cholesterol and increase of red blood-cells in the woman.

The psychosis in the woman began about fifteen years later than in the man, apparently ran a more acute course, and has progressed further. She shows a greater intellectual decay than her brother does, and, unlike him, she expresses delusions. These delusions are on the whole of quite a simple nature, and are little elaborated. Her remark that she is older in the sun than in the shade is at its face value typically schizophrenic; yet it may be the expression of a normal human striving. It is reminiscent of the attitude of the spinster in

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W. S. Gilbert's verse who "may very well pass for 43, in the dusk with a light behind her". She is Queen Annie of Hyde Park, happily married to a Mr. Algy, with many children. Perhaps these delusions over-express the wish-fulfilment dreams of an unmarried woman, cursed in the prime of life with hirsutism and obesity, whose judgment is now disordered by a psychosis. Her delusions do not occupy a prominent position in the total clinical picture. Yet their presence, and her progressive apathy and deterioration of behaviour, have led her to a diagnosis of dementia præcox. It need hardly be emphasized that the application of the term "dementia præcox" to all cases showing this classical triad of symptoms has little more significance than that of a convenient diagnostic label.

In both patients the deterioration of behaviour is marked and of the same type. There is good evidence that this deterioration is not dependent on a decay of intellect. The degree of intellectual preservation stands in contrast to the disorder of conduct. The slump of behaviour is not secondary to active withdrawal into phantasy and delusional preoccupation. The picture, especially in the man, suggests the organic rather than the schizophrenic reaction type.

The principal symptom in both cases is the marked lack of emotional drive which is seen against a background of symptoms and signs indicative of endocrine malfunction. It is to be noted that the signs of endocrine disorder in the man might easily have been overlooked. Yet the dysplastic physique, obesity, hyperpiesia, pigmentation and loss of sexual activity, though not marked, are suggestive. It is perhaps unwise to speculate on the exact nature of the endocrine disorder. I hope some day to examine the endocrine glands histologically and to report on them. The clinical evidence suggests excessive activity of the adrenals or of the anterior pituitary or of both. Although, in the case of the woman, it is known that the psychosis began with endocrine disorder, that is not evidence that endocrine malfunction is the only, or even the principal, causal factor.

Such a psychosis is uncommon, perhaps rare. Notkin found only 8 patients showing evidence of gross endocrinopathy out of 6,000 mental hospital patients, and 3 of these patients were mental defectives.

The parents were first cousins. This finding is important. A disease determined by recessive Mendelian factors is manifest only in persons who receive a recessive contribution from both parents, in whose constitutions the factor may be only latent. When the disease is rare, the carriers are uncommon. In such a case, a person who is a carrier will be more likely to meet a similar carrier among his own family than among random acquaintances. The finding of a high incidence of blood-related parents among many cases of a rare constitutional disease is pathognomonic of a single recessive gene determination. According to Mendelian laws, a proportion approaching a quarter of the children of parents, both of whom are carriers, may be expected to

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manifest the disease. The disease will have familial incidence; the parents and more distant relatives will generally be unaffected.

These conditions are fulfilled in this family. We find the familial incidence of a rare disease in the offspring of consanguineous parents. Such an association is unlikely to be due to chance. It is therefore probable that this psychosis is in part determined by a single recessive gene.

Summary.

A sister and a brother are described suffering from similar chronic, atypical and uncommon psychoses, characterized by the insidious onset of a progressive lack of emotional drive leading to gross deterioration of behaviour and secondary decay of memory and intellect, lack of insight, and, in one case, delusional formations; associated throughout the course with signs of endocrine malfunction, dysplastic physique, obesity, skin pigmentation, hyperpiesia, hypertrichosis in the woman, diminished sexual activity in the man. High blood cholesterol and increase of red blood-cells were found in the woman. The syndrome may be described as a chronic, progressive, deteriorative behaviour-slump, associated with endocrine malfunction suggestive of hyperactivity of the adrenals or of the anterior pituitary. The parents are first cousins. It is probable that this syndrome is in part determined by a single recessive Mendelian factor.

I wish to thank Dr. R. C. Turnbull, medical superintendent of Severalls Mental Hospital, for permission to publish the case-notes. I am indebted to Dr. A. Glen Duncan for cholesterol estimations, and to Dr. Aubrey Lewis for information about the literature. This work was in part done during the tenure of a grant from the Medical Research Council.

Reference.—Notkin, J., "A Clinical Study of Psychoses Associated with Various Types of Endocrinopathy", *Amer. Journ. Psychiat.*, 1932, xii, p. 331.

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Clinical Notes and Cases.

A CASE OF PROFOUND DISSOCIATION OF THE PERSONALITY.

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AND

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A BRIEF summary of this patient's condition was given in our article on "Hypnosis in Mental Hospital Practice" (1), where it was cited as Case 2, illustrating "the great value of hypnosis as a quick method of bridging amnesic gaps". The hope was there expressed that the case would be published separately in full, as it contains numerous features of general interest. Similar cases appear from time to time in the newspapers, and it seemed to us that a detailed account, showing exactly how the amnesias arose and how the case was dealt with, might be welcomed.

STATE ON ADMISSION.

The patient was admitted to Cefn Coed Hospital, Swansea, as a voluntary patient on February 22, 1936, in a semi-confused and dreamy state. She said she was single and gave her age as 20, but she was uncertain whether the number of her house was 2 or 44 in a certain terrace at G—. She gave the year as 1935, and she could not name the month. She spoke in an affected, drawling voice, saying, "I feel in an awful muddle. I can't remember when I first met my young man. I am mixing up the names and places and numbers. I cannot concentrate, and when I try to read I do not know what it is about. I feel I have been in a fog for a very long time".

She stated her father had died some weeks ago after a long illness, and that she had been very attached to him, but she could not get on with her step-mother. She also declared that the day before admission she was in a car accident which caused the death of her step-brother, but she was unable to give any details about this. Both these statements were found to be incorrect. She complained she could not sleep because her dead father came to visit her at nights. She gave a fragmentary story about a young man, B—, who had told her they were engaged and that he had furnished a house for her. It

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appeared that the car in which the accident had occurred belonged to B—, but everything was shadowy and confused. She was vague and indefinite in her account of herself and seemed to have no background.

History.

The day after her admission her stepbrother, who was supposed to be killed, came to the hospital. He knew nothing of a car accident. He reported that the patient had been working as a domestic servant for six months at Mrs. M—'s house in Swansea, and during that time she had not kept in touch with her family in G—, where her father was seriously ill, but not yet dead. The stepbrother thought this very strange, as she was known to be especially devoted to her father. About two years ago she had arrived home from London with a romantic story of being engaged to an Italian, who was to have come to arrange with her father about marrying her; but the Italian did not appear, and the father concluded there was no such man. The number of her house was 2, and not 44.

Then another young man, A—, and his mother appeared, saying that they had known her for four months. They reported that she had been staying at their house in Swansea for the past week, and had been telling some peculiar stories about her very wealthy grandmother in London. This grandmother was supposed to be coming to Swansea in a Rolls Royce car. and patient took A— round to look for a house for her. The day before admission the patient announced that the grandmother had arrived and they were all to get ready to go to her house for dinner. That afternoon, however, she disappeared and did not reappear till next day. She was very distressed, saying that her stepbrother had been killed in a car accident, and there was no further mention of the grandmother. She did not seem to know where she had been during the night of February 21. No car accident could be traced, but they all went to the General Hospital to see if her stepbrother had been taken there. At the hospital patient fainted and was seen by the house surgeon. During his cross-questioning she complained of "terrible headaches ever since she had fallen down a quarry six months ago". He recommended her to come to Cefn Coed Hospital. She had told A— early in January that her father had died and had actually gone into mourning for him.

Her stepbrother and this boy A— had not previously met, so the discrepancies in her stories were unknown to them. Only when they compared notes after her admission did they realize that something was seriously wrong with the patient. Her employer, Mrs. M—, was also seen, and reported that the girl had departed without warning, leaving a note, "I am sorry I had to do this, but I am going to a job in Cardiff". Mrs. M— had not noticed anything unusual about her during her six months' period of employment. She had had a day off some weeks previously to go to her father's funeral.

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It was also discovered that the patient's luggage was missing and neither she nor anyone else knew where it could be.

Change of Personality in Hospital.

Four days after entering Cefn Coed Hospital the patient suddenly asked where she was and how she got there. She stated she had "come out of the fog" and now did not know what had been happening while she was in the fog. She was no longer dreamy and her voice had lost its affected drawl. She now gave her age as 22, and said her family had remarked on her dropping two years some months ago. She was now a brisk and alert personality, with a good intelligence, and somewhat alarmed at the predicament in which she found herself. She was fully co-operative and did her best to straighten things out, and she showed a normal astonishment at the conflicting stories she had given.

She now knew that her father was alive, though seriously ill, and she had no memory of a car accident. When she was visited by A— and his mother she did not know them. She said, "His face seemed familiar but I did not know that I had been out with him, nor do I remember staying at his home. He says I stayed there a week". She became very concerned about her relationship with A—. She treated the matter humorously on the surface, but underneath she was deeply alarmed. She wondered whether she was engaged to this unknown man, and she did not like to ask him what had happened between them. She could not describe his house, and all memory of her employment at Mrs. M—'s house had vanished. She had in her handbag a little girl's photograph which A— told her was Mrs. M—'s daughter, and she kept studying it, but was unable to recall anything about that part of her life. She also found a house key in her handbag which she could not account for. She had no idea why she had thought her house might be number 44 instead of 2, as she had never had anything to do with a house numbered 44.

When she was questioned about the other young man, B—, she was quite frank about him, and said she had met him once in Swansea about a year ago and certainly was not engaged to him. She knew nothing about the house he was supposed to have furnished, and she was never particularly interested in him. She was amazed when she heard the story she had told about her grandmother and said, "My grandmother is a patient in a mental hospital and has been for years. How could I have said such things!" She was entirely unable to recall where she had spent the night of February 21, and still had no idea where her luggage was.

As regards the Italian, whom she had not mentioned to us before, she was now quite definite, and gave the following account: She had met him in London two years before and was very much in love with him. He had a home ready for her and a very fine car which she always said was like a Rolls

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Royce. When, however, she announced her engagement to her parents, they objected, and her father in particular was very upset about it. As she was so devoted to her father she developed a conflict, especially as her father was seriously ill with cancer. The Italian was to have come down in August, 1935, to see her father at G—, but when she refused to become a Catholic they quarrelled and he did not come. She had not seen him since, and did not know of his whereabouts at the present time.

On the fifth day after admission her visitors told her that her father had died. She took this very well and wrote a sympathetic and quite affectionate letter to her mother on the subject. It was found she had no memory of her father appearing to her at night.

Method of Investigation.

The results in the case to date were now presented and discussed at a staff clinical meeting, where it was established that her amnesia began early in August, 1935. She remembered quite clearly "going out very worried for a walk" after quarrelling with her father over the question of getting married to the Italian; but "since then my head has been funny; I'd go out for walks and not know where I'd been". It was decided to investigate the case under hypnosis and try to fill up the blanks in her memory.

Hypnosis was induced with exceptional ease, and she passed readily into the deepest stage in which negative hallucinations could be produced. She was encouraged to fill up the gaps since August, 1935. The material she produced was extensive, but always to the point. She had five sittings of about one and a half hours each under hypnosis, during which she steadily recounted all she could remember of her movements in chronological order. We took notes of what she said, and next day cross-questioned her in the waking state on the previous day's revelations. Each day she still had occasional small gaps in the recollections recovered the previous day, and these were cleared up under hypnosis before proceeding further with her story. Hypnosis was induced by both of us, and the "rapport" could readily be transferred from one operator to the other as desired. In this way, by exploration in hypnosis, she eventually recalled almost *in toto* the events that had occurred during her six to seven months' amnesia.

It was found that she had been subject to a series of extensive fugues almost constituting a continuous fugue for the whole period. When, under hypnosis, she came to points in her story where she changed from one fugue sequence to another, she complained of momentary severe headaches at each change-over. In the early part of the story the headaches were occasional, but they gradually increased in frequency, and from her account of events in February, 1936, she seems to have had almost daily alternations of her personality, till the strain of events was lifted from her by admission to hospital.

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FINDINGS UNDER HYPNOSIS.

It was found that the amnesia was precipitated by a fall while in a highly emotional state, and was kept in operation by a resulting tangle of events from which she could not escape. When she went for the walk early in August after quarrelling with her father, she came to a quarry and heard a dog whimpering on a rock ledge below. She let herself down by a branch to the ledge to rescue the dog, but she slipped and fell some distance and seems to have been slightly concussed (evidence of first headache shown at this point). A week later she answered an advertisement and went to a post at Mrs. M—'s. At this stage she "forgot all about the Italian and my father", and in October she met the boy A— casually and became friendly with him. They were both keen cyclists and went out a good deal together. Later she was invited to his house on her days off duty and he fell in love with her. He told her this, but she "did not feel that way about him", and she "said he was not to say it again". Then she was reminded of her father by a letter from her mother (another headache). She went home to see him and found him very thin and changed. Her stepmother upbraided her for staying away so long. She tried to tell her stepmother that she had forgotten about her father, but she could not explain "it", and her stepmother said, "Don't be silly; how could you forget him?". She went back to Swansea (headache) and said at this point, "I had another bad head and forgot all about home again".

The other boy, B—, was now in Swansea and he began to cause her trouble. He, too, said he loved her and wanted to marry her. She did not like him particularly and quarrelled with him in order to discourage him, but the more she quarrelled with him the more he importuned her and she began to get frightened as she could not shake him off. Early in February B— said he had taken a house and was going to furnish it for her, and he persuaded her to go and look at it with him. He gave her the key and she put it in her bag. The house was No. 44. They had a violent quarrel because she still would not marry him, and she ran out of the house with the key in her handbag (more headaches at this point).

She now thought her father was dead and that she ought to leave Mrs. M— and go home to comfort her mother. At this time she "saw" her father every night when she was in bed, and she could not sleep because he kept "talking" to her. Then, a week before admission, the boy B— came round to see her at her place of employment, so she decided definitely she must leave Swansea, and she "got more bad heads". The next day Mrs. M— was out and patient suddenly decided to go to Cardiff. She said under hypnosis, "My mind was again not my own". When she went up to get her suitcase she found she had already packed her clothes, so she left immediately. At the station she had to wait an hour and a half for the next train to Cardiff, and the ticket-collector offered to mind her suitcase while she got some food in a

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shop across the road. " In the shop I came to myself again and did not know what it was I had been intending to do." So she went to A—'s house. She could not explain there what she had done because she did not know. A—'s mother asked her to stay, and she remained there all the week, but there were a lot of headaches.

On February 21 (the day before admission) she went into the town from A—'s house to get her hair waved and accidentally met B— again. This upset her so much that her " head felt too bad " to get her hair done, and she went back to A—'s house in a thoroughly muddled state, intending again definitely to leave Swansea. Her recollections of this period under hypnosis were not as complete and orderly as all the previous part of her story had been, and she kept interrupting her account with storms of headaches and rapid changes of outlook as first one personality and then the other took up the story. It seems she left A—'s house in the evening to go and say good-bye to Mrs. M—, but instead she wandered about the country round Swansea all night. She arrived outside Mrs. M—'s house about 7.30 a.m. on February 22 (the day of admission), and at this point she said, " I suddenly realized I didn't know what I was doing, so I did not go in ". She then thought she would throw herself under a car and end it all. Instead she drifted to B—'s house, and still having the key, went in and sat down. B— found her there, and there was more quarrelling. Eventually he took her for a ride in his car. When he was driving fast near A—'s house she saw a child in the road, and as he took no notice she caught hold of the steering-wheel and the car ran into a wall. She thinks she must have knocked her head against the top of the car, and that B— was bleeding from a cut, but not seriously hurt. She remembers thinking that B— was her stepbrother, but is quite confused about how she got to A—'s house again. She remembered being taken to the General Hospital to see if her stepbrother was there, and she found the building unknown to her though she should know it quite well, having frequently visited her father when he was a patient there before her troubles began.

She was in such a vacillating, confused state in the last twenty-four hours before admission that she was not able to recall all she had thought and done. One illustration of this is her story about her grandmother. As has been already mentioned in the history, her grandmother is a patient in a mental hospital, and just before she left Mrs. M—'s post the girl received a letter from the grandmother asking for a visit. She explains, " I was in such a muddle I thought my grandmother was coming to see me, and the Italian's car was like a Rolls Royce, so I thought she was coming in that ".

Further Progress.

The gaps in her memory were thus almost completely filled up, and explanations of the psychogenic nature of her illness and its causes were given to her in the waking state. She was intelligent, and readily accepted these after

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putting some shrewd questions about the workings of her mind. She behaved normally in the ward and remained quite well till just before her first week-end at home, when she became temporarily unsettled as discussed below. She was discharged from hospital on March 22, and two weeks later called at the hospital to report her progress. She now appeared poised and well-balanced and was arranging to take up a post again from home, as she did not get on well with her stepmother. The result is, up to the present, complete recovery, and the patient is leading a happy, useful life.

Discussion.

Where this patient's story, as given under hypnosis, could be checked it was found to be correct. A striking example of this was the resulting recovery of her luggage from the railway station, where it had been left exactly as she described. The car accident could not be verified, and here we feel some difficulty. The occurrence of the accident so near to A—'s house seems to be stretching coincidence too far. If it did occur as she described it her continued vagueness about it might be accounted for by a superimposed traumatic confusion, but in that case why was she allowed to find her own way to A—'s house alone? On the whole one feels that the accident did not occur, and that here hypnosis tapped a phantasy arising either out of her admitted confusion on that day, or purposely elaborated to divert A—'s attention from her inexplicable absence all night. It is regretted, for the sake of completeness, that the boy B— was not interviewed. He undoubtedly existed, because her relatives knew him, but it was felt advisable that he should lose all touch with her whereabouts.

The change in her personality when she "came out of the fog" four days after admission was most striking and definite. While "in the fog" she was dreamy and silly, and could easily be labelled an hysterical person. After the sudden change she showed no further hysterical manifestations except at the appropriate places in her recital under hypnosis. She was normally a level-headed girl who knew her own mind, and was most unlikely to have got herself into the tangle of events she related. There is no doubt she was no longer in effective control of her actions after her fall down the quarry. The dissociation then produced led to difficulties that were dealt with inadequately by further dissociation. She frequently used words in her narrative which showed that she appreciated her difficulties and wanted to run away from them: "Here am I giving up the Italian for my father and God has taken my father from me. How I wish I could be someone else to please my mother! There was my father and the Italian. There was B— and A—. I just did not know what to do. I thought I would just go away and leave them altogether."

The underlying temperamental defect in her character was revealed before her discharge when it was arranged that she went home for a week-end to test

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herself. As the time approached she lost her poise and became upset, obviously distrusting her ability to meet the situation, even though she knew the facts had been explained to her relatives. She tried to evade the issue till firm pressure was put on her to make her go. She returned confident, and her relatives reported they found her more normal than she had been for many months. This disinclination to face facts was specially pointed out to her, and its general bearing on her troubles was made clear to her. She was a strongly extroverted personality in sudden acute conflict with a father whom she adored, and a stepmother with whom it is known she could not get on. She was remarkably easily hypnotized and passed rapidly into the deepest stage, in which a single suggestion that there was no such letter as Y in the alphabet resulted in her writing her name with a blank space for the omitted Y. It is obvious that such a personality is easily dissociated. So the shock of falling in the quarry while in a state of emotional turmoil was sufficient to initiate an amnesia. The disinclination to face facts was the fundamental underlying cause, and this allowed the dissociation to remain and increase its scope. In this connection it ought perhaps to be mentioned that nothing she need be ashamed of had occurred between her and the men involved in her story.

As McDougall (2) points out in his *Outline of Abnormal Psychology*, fugues, somnambulisms, hysterical fits and alternating personalities are all manifestations in varying degrees of the same fundamental psychopathological process, namely, a tendency to dissociation. This dissociation may be slight, involving only perhaps a limb, and will then manifest itself as a conversion symptom, or may result in a profound cleavage of the entire personality, so that two or more subsidiary personalities may alternately take possession of the self-regarding sentiment. This patient would appear to have had a dissociation of marked degree which, if untreated, might even have progressed further.

We are indebted to the Medical Superintendent, Dr. N. Moulson, for permission to publish this case.

References.—(1) Copeland and Kitching, "Hypnosis in Mental Hospital Practice", *Journ. Ment. Sci.*, 1937.—(2) McDougall's *Outline of Abnormal Psychology*.

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Part II.—Reviews.

Convulsion Therapy in Schizophrenia [Die Konvulsionstherapie der Schizophrenie]. By LADISLAUS VON MEDUNA. Halle a/S.: Carl Marhold, 1937. Pp. 121. Price RM. 4.00.

In this book Dr. Meduna, who is Superintendent of the Royal State Mental Hospital in Budapest, gives an account of the convulsion treatment by means of intravenous injections of cardiazol, of which he is the originator. Altogether 110 patients have been treated, and 74 pages are devoted to a survey of the 54 remitted cases. Full details of their previous history and response to treatment are given, but information regarding their after-history is scanty.

Details of the theory and technique of the treatment will be found elsewhere in this issue of the Journal. It is difficult to say how many convulsions must be induced to secure a remission. In several cases two to three were sufficient; others again required twenty-five to thirty. The full course takes from six to eight weeks, though in some cases it may take considerably longer. As a general rule, treatment is not stopped until fifteen to twenty convulsions have been induced. Experience has shown that even after complete remission has set in, at least three further convulsions must be induced, to guard against the possibility of a relapse. It is not easy, however, to persuade patients, in particular those progressing well, to submit to subsequent injections. They come to dread these, and implore the doctor to let them off. Difficult as it is to resist their entreaties, such temptation must, for the patient's sake, be resisted, as treatment has to be continued well into the remission stage to prove effective.

Generally speaking, the more readily patients react to a small or average dose, the more likely are they to remit. After four to five convulsions have been induced, it is usually possible to form some opinion as to whether the patient will or will not improve. Remission may either take place suddenly, without an intervening period of lucidity, or gradually; in some cases it is of an intermittent type. Strangely enough, those remitting suddenly do not appear to be elated over the fact, but take it rather as a matter of course. Very little is said of psychological phenomena occurring during treatment and remission.

Treatment does not appear to be unduly dangerous. No deaths or serious complications occurred in the 110 cases under review, in which over 1,000 convulsions were induced. Transient attacks of moderate tachycardia were observed, and twice the arm and twice the lower jaw dislocated. Renal complications did not occur. In the opinion of independent observers, the heart and circulation did not suffer from the treatment. Needless to say, the therapy is contra-indicated in all cardio-vascular diseases.

In the course of treatment the blood-picture shifts to the left, the relative number of neutrophile leucocytes being increased and that of the lymphocytes

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reduced. This phenomenon contains a certain prognostic value, as it is much more pronounced in remitting patients. The author believes that the urinary changes are similar to those produced by genuine epileptic attacks. After the convulsions, traces of lactic acid were found in every case. On the average, acidity is increased by 26%, the ammonia content by 14%, the phosphate content by 10% ; the chlorides decrease by 25%.

In presenting his results, the author refrains from quoting percentages, as in his opinion the number treated is insufficient for this purpose. He distinguishes only between cases of remission and of non-remission. Of the 110 patients under review, 54 remitted. Very little is said of what in the author's opinion are the criteria of remission. He is difficult to follow when he adopts three different ways of discussing his results. Firstly, he arrives, it would seem on *non lucendo* principles, at the conclusion that all patients who remitted belong to a group of " symptomatic schizophrenia ", while those failing to remit are placed in a group of " endogenous schizophrenia ". In the latter treatment is hopeless, no matter how short the duration of illness. Secondly, what might be called the anatomical phase of the disease plays an important role. Dr. Meduna distinguishes between three phases of schizophrenic disease : (*a*) The " acute process ", represented by the initial onslaught of the disease ; (*b*) the " process ", i.e., the disease still going on, though the first onslaught is over ; (*c*) the " post-process ", in which the disease has come to an end, leaving defects behind. Arranging his cases under these headings, he finds that in phase (*a*) 39 of 41 patients remitted ; in phase (*b*) 12 of 21 ; and in phase (*c*) only 3 of 48. He concludes that so far as so-called process-schizophrenia is concerned (represented by phases *a* and *b*), 82% remissions can be obtained, always on the understanding that such patients belong to the symptomatic variety. Lastly, the results are given according to the duration of illness. The author himself now prefers this way, which incidentally allows for comparison with the results of other forms of treatment. It was found that the following cases did not remit : Duration of illness under ½ year, 3 of 36 patients ; under 1 year, 1 of 7 ; under 1½ years, 4 of 7 ; under 2 years, 5 of 11 ; under 3 years, 9 of 12 ; over 3 years, 34 of 37.

Dr. Meduna believes that convulsion therapy is only the first step on a biological road of influencing schizophrenia. Far from holding that his method is the only efficient therapy, he advocates—and those experienced in both forms of treatment will readily agree—that convulsion therapy and insulin therapy should not strive to outdo one another, but should rather reinforce one another in combating the disease. H. PULLAR STRECKER.

The Growing Child and Its Problems. Edited by EMANUEL MILLER. London : Kegan, Paul, Trench, Trübner & Co., 1937. Price 6*s*. net.

This volume adds another to the growing list of short books, written in a more or less popular style for the more or less informed public. The essays are contributed by five psychiatrists, all of whom can claim an unusually wide experience in the handling of problem children. None represents any particular school to the exclusion of all others, so that, as a general survey of the field of child psychiatry, the cases encountered, and the methods in use, this volume supplies a need.

But, as so often happens, for the lay reader one would imagine that such an essay as Dr. Allen's on " Personality Deviations " might prove perplexing ;

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while to the expert, others may do little more than repeat what must be common knowledge. So long as so wide a field is covered in 230 pages, it is certain that much will perforce be dealt with in a very superficial way.

Nevertheless, a sound attempt is made here to cover the field. The psychologist's role, both as to play material and educational assistance, is excellently described by Miss Chesters and Miss Simmins. The psychiatrists deal with problems of adolescence, in girls by Dr. Laura Hutton, and in boys in an admirable essay by Dr. Miller. Dr. Paterson Brown's essay on "Habits" is particularly clear and concise, and is really written about children rather than about child psychology. Neuroses in school-children by Dr. Burns discusses points in practical methods and therapy, and Dr. Allen discusses personality deviations from the Freudian standpoint.

The essays would certainly afford an interesting basis for a series of lectures, followed by, and serving to introduce, discussion, but no indication is given that this was, in fact, their purpose originally. One is thus left with a somewhat haphazard impression as to how much can be learnt and understood as a result of reading this book. E. M. CREAK.

Reading, Writing and Speech Problems in Children. By SAMUEL T. ORTON. London: Chapman & Hall, Ltd., 1937. Price 10s. 6*d*. net.

Dr. Orton's work on the nature of reading, writing and speech problems in children deserves to be more widely known in this country than it is. Fortunately the occasion of the Salmon Memorial Lectures has given Dr. Orton an opportunity to collect and consider the outcome of his unrivalled experience in this field.

Dr. Orton has never deviated from his conception of the reading difficulty as part of an impairment in the development of the language function as a whole. His researches have done much if they have served to draw attention to the association sometimes of such problems as left-handedness and stammering, and mixed dominance and a failure to learn to read. All who are familiar with these cases in practice know how correct is his observation that one of the major difficulties experienced by these children is in the correct linkages of letter sequences in a word, whether read silently or spoken aloud, and his theory offers a comprehensive explanation, both for the group as a whole and the individual variations within the group.

No good, however, will be done by a theory which is valid only by assumptions. The physiological interpretations of the significance of cerebral dominance are more intriguing than they are convincing. Nevertheless, the spirochæte was suspected many years before it was found in the cortex of the general paralytic; perhaps some Noguchi of the future may succeed in locating the engram. While Dr. Orton has clearly withdrawn those assumptions which have proved untenable, it must be admitted that his conception, while clinically acceptable, is still a big pill for the cerebral physiologist to swallow. What he has undoubtedly added to our knowledge is the recognition of the syndrome of developmental language difficulty as a whole. A research based on ten years' painstaking work on nearly 1,000 cases cannot be lightly dismissed.

In discussing treatment, Dr. Orton is a convinced advocate of the phonetic method of teaching reading. It remains a fact that in skilled hands, even for the strephosymbolic, the "look and say" method may give excellent results in coaching and re-training. Possibly it is as true in this field as in

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others in clinical psychiatry, that what is said to a child matters less than the way in which it is said, and it is a welcome addition to this work that in this volume due weight is given to the importance of emotional factors, acting in a child predisposed to such a language difficulty.

It is to be hoped that a yet fuller work will come from Dr. Orton's department. Freed from the necessity of describing his work in the form of spoken lectures, it is hoped that his next book will include a bibliography and an index, both conspicuously lacking in the present volume.

E. M. CREAK.

On Suicide [Der Selbstmord]. By R. WEICHBRODT. Basel, 1937. Pp. 252. Price 19 Swiss francs.

There is an excellent and comprehensive German bibliography of the gigantic literature on suicide, compiled by Rost a few years ago ; but an up-to-date monograph on the subject has so far been missing. The book of Dr. Weichbrodt fills this gap. He tries to steer a middle course between a popular introduction and a scientific review of our present-day knowledge. In spite of the difficulty inherent in this course, the author succeeds in covering every possible aspect of suicide without piling up too much detail. The clear and unpretentious style makes the book especially pleasant to read.

Although a physician by profession, Weichbrodt devotes a considerable amount of space to suicide in its historical aspect and from the point of view of literature. The appendix, forming a quarter of the book, does not consist, as one might have expected, of statistical tables and diagrams, but contains the life-histories of five famous Germans who committed suicide, and writings on the subject by celebrities from Flavius Josephus to Schopenhauer.

The first part of the book deals with the history of suicide in different ages, religions and nations. A chapter on suicide and superstition concludes this part. The ideas of philosophers and poets on the subject are given in interesting and learned quotations. The facts on " suicide and law " are given in a short but comprehensive survey.

The second half of the book starts with statistics. Problems arising from the statistical methods and results are thoroughly discussed. A special chapter is devoted to suicide of soldiers, and the relation of suicide to mental abnormality is treated with expert knowledge. " Methods of suicide ", " motives ", " flirtation with the idea of suicide ", and " suicide and love " are treated from a psychological angle. Dr. Weichbrodt's bird's-eye-view ends with a discussion on the influence of economics on suicide. In his conclusions he points to the relative incompleteness of our present knowledge, and emphasizes the difficulties in the way of prophylactic measures. W. MAYER-GROSS.

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INDEX TO VOL. LXXXIII.

The General Index covers original articles, reviews and the epitome, and includes subjects dealt with as well as the titles of articles.

The following names are printed in SMALL CAPITALS: authors of original articles and of books reviewed. Names of authorities referred to in the text are in ordinary type.

(R.) indicates a *Review*; the title of the book reviewed is followed by the author's name, thus: "Psycho-analysis and its Derivatives, by H. Crichton-Miller (R.)."

(E.) indicates the *Epitome* section; the title of the abstract is followed by the author's name, thus: "Age and human ability. Miles, W. R. (E.)."

The names of authors referred to in the Epitome section are listed in Part II.

PART I.—GENERAL INDEX.

Acetarsol in the treatment of late congenital syphilis amongst mental defectives, by K. C. L. Paddle, 372
,, toxic effects of, 391
,, treatment by, hæmatology in, 387
Acetylcholine, action of, on carotid sinus. Heymans, C., *et al.* (E.), 238
,, and curarine, antagonism between. Briscoe, C. (E.), 123
,, and eserine, related compounds, action of, on central nervous system (E.), 604
,, and other constituents of adrenal gland, effect of, upon blood sugar and amino-acids. Davis, B. L., jun., and Luck, J. M. (E.), 244
,, breakdown in blood and cerebro-spinal fluid. Altenburger, H. (E.), 597
,, central action of. Silver, G. A., and Morton, G. (E.), 122
,, formation of, by brain tissue. Stedman, Edgar, and Stedman, Ellen (E.), 593
,, in chemical mediation of nerve impulses. (E.), 132
,, ,, transmission of nervous influx through central synapses (E.) 132
,, in traumatic epilepsy. Fribourg-Blanc (E.), 131
,, intracarotid injection of, in man. Battro, A., and Lanari, A. (E.), 239
,, is the action of vasodilator nerves due to liberation of? Tournade, A., and Chevillot, M. (E.), 584
,, liberated from a complex substance in nerve centres. Corteggiani, E., *et al.* (E.), 344
,, liberation from stimulated nerve of sensitizing substance. Bergami, G., *et al.* (E.), 333
,, -like substance, liberation of, from cut surface of nerve. Bergami, G. (E.), 231
,, ,, substances, parasympathetic stimulating and paralysing actions of. Blankart, A. (E.) 339
,, mechanism of a sympathetic ganglion. Brown, G. L., and Feldberg, W. (E.), 245
,, ,, of biological synthesis of. Stedman, Edgar and Ellen, 595
,, of brain, free and combined. Corteggiani, E. (E.), 593
,, role of, in transmission of nervous impulses through a sympathetic ganglion (E.), 582
,, ,, ,, ,, stimulation in striated muscle. Lapicque, L. (E.), 593
,, vagal substance of Loewi and Viale's vagotropin, relations between. Bartolini, B. (E.), 128
Acetyl-β-methylcholine (mecholyl) and its relationship to other drugs affecting the autonomic nervous system. Myerson, A., Loman, J., and Dameshek, W. (E.), 238
,, ,, physiological effect of. Myerson, A., Loman, J., and Dameshek, W. (E.), 603

Digitized by Google

Acrocyanosis, by E. S. Stern, 408
,, disorders related to, 428
,, mechanism of, 415
,, pathology of, 422
Acromegaly, insulin response in (E.), 691
Administrative Psychiatry, by W. A. Bryan (R.), 571
Adrenaline, action of, on cerebral vessels (E.), 231
,, ,, on the knee-jerk. Schweitzer, A., and Wright, S. (E.), 339
,, and sympathin, liberation of, induced by stimulation of hypothalamus. Magoun, H. W., Ranson, S. W., and Hetherington, A. (E.), 584
,, effect of, on vasomotor centre (E.), 103
,, effects of, on cerebral circulation. Cachera, R., and Fauvert, R. (E.), 122
,, secretion, effect of injury of cerebellum on (E.), 103
,, ,, nerve centres controlling, in high cervical region of dog. Jourdan, F. (E.), 105
Affective psychoses, observations on endocrines in, 52
Agoraphobia, case of. Greenschpoon, R. R. (E.), 234
Agranulocytosis, relative to barbiturates (E.), 600
Alcohol addict, ætiology of polyneuritis in (E.), 111
,, concentration and excretion of, in normal, pellagric and alcoholic subjects. Tomesch, P., and Dimolescu, A. (E.), 345
Alcoholism and vitamin B deficiency, experimental study of. Wechsler, I. S., Jervis, G. A., and Potts, H. D. (E.), 111
Alkalosis, in relation to spontaneous hyperventilation tetany, 199
Allen, A. H. B., The Self in Psychology (R.), 575
Alzheimer's disease, relative to Pick's disease and to senile dementia (E.), 589, 590
Anæmia, neurological and psychotic syndromes with. de Morrier, G. (E.), 336
Anæsthetics, brain content of, in experimental states. Tiffeneau, M. (E.), 240
Anatomy and Physiology epitomes, 103, 229, 333, 478, 582
Anatomy, Physiology and Hygiene: A Text-book for Nurses, by A. Millicent Ashdown and E. Bleazby (R.), 330
,, ,, First Aid and Hygiene: A Preliminary Handbook for Nurses (issued by the Department of the Interior, South Africa) (R.), 330
Anencephalons and their behaviour. Sokolanski, G. G. (E.), 584
Aneurysm of intracranial portion of internal carotid artery, syndrome of unruptured. McKinney, J., Acree, T., and Soltz, S. E. (E.), 114
Anoxæmia, in relation to gliosis in mental defectives, 262, 264
Anthropometry in manic-depressives and schizophrenics, 56
Anxiety, psychobiology of. Yuskin, J. C. (E.), 585
Aphasic subjects, song and re-education in. Baldi, F. (E.), 125
Arsenic and lead, some neurological signs produced by. Cornwall, L. H. (E.), 116
Art and therapy in the mental disturbances of children. Bender, L. (E.), 606
Arterio-sclerosis, hypertension and heart failure, psychoses associated with, 551
Ascaris infestation and infantile paralysis. Martini, F. (E.), 337
Ashdown, A. Millicent, and Bleazby, E., Anatomy, Physiology and Hygiene: A Text-book for Nurses (R.), 330
Atropine in the cure of post-encephalitic parkinsonism. Budinis, I. (E.), 240
Attis, a study of castration. Eagle, B. S. (E.), 234

Barbital and phenobarbital, mechanism of action on vegetative nervous system. De Biasio, B. (E.), 600
,, ,, relation of, to granulocytopenia. Kopet, J. C., and Goodrich, F. J. (E.), 600
,, derivatives, toxicity of. Jones, I., and Lynn, E. V. (E.), 119
,, new method for determination of. Sack, G. (E.), 600
,, sodium, delayed death and mortality following cardiazole and ephedrine after lethal doses of. Gros, O., and Hofmann, H. (E.), 605
Barbitone, sodium, excretion of, in patients suffering from mental illness, by E. H. Kitching and A. Dignam, 40
Barbiturate and strychnine: urine of animals after separate injections of. Lavergne, V. de, *et al.* (E.), 119
,, -picrotoxin antagonism. Koppanyi, T., *et al.* (E.), 241
,, poisoning, ephedrine as antidote to (E.), 600

Digitized by Google

Barbiturates: action of phenobarbital on adrenaline-sensitive vaso-dilators. Raymond-Hamet (E.), 601
,, alleged anticomplementary anticoagulant action of. Mollari, M., Randall, W. A., and Koppanyi, T. (E.), 338
,, comparative effects of sodium and magnesium salts of pentobarbital. Taylor, W. F., and Lackey, R. W. (E.), 122
,, effect of prolonged chloroform anæsthesia on duration of action of. Koppanyi, T., Dille, J. M., and Linegar, C. R. (E.), 240
,, effects of pentobarbital on rabbit. Blackberg, S. N., and Hrubetz, M. C. (E.), 121
,, in blood, determination of, by new method. Brundage, J. T., and Gruber, C. M. (E.), 599
,, newer, pharmacological action of. Gruber, C. M. (E.), 601
,, pathological changes following poisoning by. Nakamura, T. (E.), 338
,, peripheral activity of. Koppanyi, T., *et al.* (E.), 241
,, study of pernocton. Mannini, R. (E.), 721
Barbituric acid derivatives, nitrogen-alkyl. Swanson, E. E. (E.), 239
Barbiturism, acute, of hallucinatory and pseudo-cerebellar type. Euzière *et al.* (E.), 601
Barbiturism, chronic experimental. Carratala, R. E. (E.), 120
Belief, The Psychology of, by H. Lundholm. (R.), 578
Bellergal, effect of, on choline esterase level, 205
Benjamin, A. Cornelius, The Logical Structure of Science. (R.), 575
Benzedrine, central action of. Nattanson, M. H. (E.), 238
,, pharmacology of. Detrick, L. E., *et al.* (E.), 598
,, physiological effects of, and relation to other drugs affecting autonomic nervous system. Myerson, A., Loman, J., and Dameshek, W. (E.), 238
,, sulphate, in post-encephalitic parkinsonianism. Solomon, P., Mitchell, R. S., and Prinzmetal, M. (E.), 484
,, ,, stimulating action of. Davidoff, E., and Reifenstein, E. C. (jun.) (E.), 484
,, , use of, in catatonic stupors. Carlisle, C. L., and Heckel, C. H. (E.), 341
,, treatment of narcolepsy with. Tihen, H. N. (E.), 599
Berger rhythm in epilepsy, 137
Bile-salts, influence of, on nervous system. Lichtman, S. S., and Stern, E. L. (E.), 128
Biological Time, by Lecomte du Noüy (R.), 97
Biology of social life, the, by Sir W. Langdon-Brown (Maudsley Lecture), 1
Bleazby, E., *see* Ashdown, A. M.
Blood-brain barrier: significance of electrical charge and S-potential. Friedemann, U. (E.), 486
Blood, mineral composition of, of a dog deprived of cerebral hemisphere, etc. Abuladze, K. C., *et al.* (E.), 104
,, platelets, origin of, discussion on, 176
,, ,, the enumeration of, in mental disorders, by D. K. Bruce, 175
,, pressure, effect of injury of cerebellum on (E.), 103
,, ,, low, postural (E.), 110
,, ,, *see also under* Hypertension
Blood-serum of insane, iso-electric points of. Tomioka, T. (E.), 129
,, toxicity in psychoses. Wahlström, G. (E.), 594
Boarding-out of mental patients, 489
Bok, S. T., A Quantitative Analysis of the Structure of the Cerebral Cortex. (R.), 98
Brain and other tissues, oxidation of aliphatic amines by. Pugh, C. E. M., and Quastel, J. H. (E.), 595
,, carbohydrate metabolism of: glycogen in nerve tissue. Kerr, S. E. (E.), 243
,, ,, ,, of, origin of lactic acid. Kerr, S. E., and Ghantus, M. (E.), 345
,, extracts, autolysis of, formation of inorganic phosphoric acid during. Gorodisskaya and Epshtein, S. (E.), 593
,, Frontal Lobes, Intellectual Functions of (R.), 575
,, iron, copper, and manganese content of, 452
,, metabolism and glycerophosphoric acid. Johnson, R. E. (E.), 487
,, ,, effects of hydroxymalonate on. Jowett, M., and Quastel, J. H. (E.), 594
,, ,, formation of succinic acid. Weil-Malherbe, H. (E.), 594
,, ,, identity of the indophenol-reducing substances in brain tissues. Young, F. G. (E.), 596

Digitized by Google

Brain metabolism, pyruvic acid oxidation. Peters, R. A. (E.), 596
,, ,, studies in. I. Metabolism of glutamic acid in brain. Weil-Matherbe, H. (E.), 488
,, ,, *see also under* Enzymes
,, phosphatase. Giri, K. V., and Datta, N. C. (E.), 346
,, phosphorylation and oxido-reduction of glucose decomposition in. Adler, E., Euler, H. v., *et al.* (E.), 592
,, proteins of, arginine content of. Budanova, A. M. (E.), 596
,, respiration, catarotulin effect and vitamin B_1 (E.), 592
,, ,, effect of dichlorodiethyl-sulphone on (E.), 133
,, ,, of. Dixon, T. F., and Meyer, A. (E.), 487
,, selective regional vulnerability of, and its relation to psychiatric problems. Meyer, Alfred, (E.), 109
,, surviving, pigeon's, lactacidogen and lactic acid in. Gorodisskaya, H., and Epelbaum, S. (E.), 593
,, vascular disturbances in, histopathological criteria of, 509
,, vascularity of different areas of, 510
Brain-weight–body-weight relations, effect of growth hormone on (E.), 230
BRICKNER, R. M., Intellectual Functions of the Frontal Lobes (R.), 575
Brilliant vital red as an anticonvulsant. Cobb, S., Cohen, M. E., and Ney, J. (E.) 603
Bromine, action of different doses of, on inhibition. Kaminski, S. D., and Maiorov, F. D. (E.), 604
,, administration of small doses of, to neurotic patients. Vainberg, I. S., and Birman, B. N. (E.), 603
,, blood, in manic-depressive psychoses. Bergonzi, M. (E.), 128
,, compound, aliphatic, causing nystagmic head deviation (E.), 603
,, content of cerebro-spinal fluid in nervous disease and insanity. Nagy, Mihaly, and Straub, J. (E.), 344
,, picture in blood of psychotics, new researches on. Meier, C. A., and Schlientz, W. (E.), 485
BRUCE, D. K., the enumeration of blood-platelets in mental disorders, 175
BRYAN, W. A., Administrative Psychiatry (R.), 571
Bulbar paralysis, hereditary progressive. Vialetto, E. (E.), 110
Bulbocapnine, effects of, on peripheral vascular system. Leese, E. C., and Fogelberg, A. (E.), 606

Calcium and protein changes in serum during sleep and rest without sleep. Cooperman, N. R. (E.), 132
,, antagonism of, to curarizing agencies (E.), 333
,, content of cerebro-spinal fluid in experimental tetany. Siwe, S. (E.), 134
,, ,, of different parts of brain of normal and thyreo-parathyroidectomized rats. Farago, S. (E.), 346
,, deposition of, in brain. Shimada, S. (E.), 344
,, in blood in a case of spontaneous hyperventilation tetany, 193
,, role of, in spontaneous overbreathing tetany (E.), 130
CAMPBELL, A. C. P., the relative vascularity of different areas of the mammalian brain (abstract), 510
Capgras's syndrome (E.), 336
Capillary blood-stain method of Pickworth, use and results of, 515, 519
Carbohydrate metabolism of brain (E.), 243, 244, 345
Carbon dioxide, effect of, on vasomotor centre (E.), 103
Cardiazol, action of (E.), 123
,, as antidote to barbiturates, 605
,, chemistry and pharmacology of, 611
,, coramine and methylimidazole, comparison of stimulating and restorative properties of. Axmacher, F. (E.), 605
,, treatment of schizophrenia, 610, 727
,, ,, ,, technique and results, 616
Carotid sinus, action of acetylcholine on. Heymans, C. (E.), 238
,, ,, reflex causing syncope and convulsions (E.), 236
Castrated dogs, higher nervous activity of. Arkhangelskii, V. M. (E.), 342
Castration, study of (E.), 234
Catarotulin effect, experiments on. Westenbrink, H. G. K., and Polak, J. J. (E.), 592

Digitized by Google

Catatonia, experimental, chronic, produced by indolethylamine. Nieuwenhuyzen, F. J. (E.), 345
,, ,, urethane (E.), 103
Catatonic stupor, action of benzedrine on (E.), 341
Central nervous system, evolution of, 3
,, ,, ,, metabolism of: normal respiration and influence of electrolytes. Chang, T. H., and Tai, F. I. (E.), 330
Cerebellar peduncle, superior, lesions of, effects of. Ferraro, A., and Barrera, S. E. (E.), 115
Cerebellum, effect of injury of, on blood-pressure, blood sugar and adrenaline secretion. Wada, M., Seo, M., and Abe, K. (E.), 103
,, lesions of, causing myoclonus (E.), 116
Cerebral circulation and psychosis, discussion on, 505
,, ,, effect of adrenaline on (E.), 722
,, ,, some new points in its anatomy, physiology and pathology. Putnam, T. J. (E.), 478
,, ,, some remarks on the physiology of, by F. L. Golla, 505
,, cortex, abnormal foci in, in epilepsy, as shown by electro-encephalogram, 138
,, ,, effects of urethane on sigmoid area of (E.), 103
,, ,, in relation to the thalamus (E.), 229
,, ,, Quantitative Analysis of the Structure of the, by S. T. Bok (R.), 98
,, function, tests of, in organic mental disorder, 440
,, hæmorrhage, conceptions and new researches on. Frommel, E., and Herschberg, A. D. (E.), 129
,, hemisphere, dog deprived of one, and of motor cortex of the other, composition of blood of (E.), 104
,, ischæmia and mental disorder, by F. A. Pickworth, 512
,, lesions in uncomplicated fatal diabetic acidosis. Dillen, E. S., Riggs, H. E., and Dyer, W. W. (E.), 109
,, tumour, occlusion of aqueduct of Sylvius by (E.), 114
,, ,, significance of incomplete homonymous hemianopia in. Johnson, T. H. (E.), 111
,, tumours, angioblastic meningiomas. Wolf, A., and Cowan, D. (jun.) (E.), 113
,, ,, clinical diagnosis of tumours of corpus callosum. Cramer, F. (E.), 117
,, ,, differential diagnosis of parasagittal gliomas and meningiomas. Masson, C. B. (E.), 112
,, ,, hearing in patients with. Northington, P. (E.), 112
,, ,, neoplastic cysts communicating with lateral ventricles. Cohen, I. (E.), 117
,, ,, origin of increased cerebro-spinal fluid protein in (E.), 245
,, ,, prolonged fever following removal of, from posterior cranial fossa. Ehrlich, W. (E.), 482
,, ,, roentgen treatment of, by direct radiation. Elsberg, C. A., Davidoff, L. M., and Dyke, C. G. (E.), 482
,, ,, tumours at base of brain, their relation to pathological sleep. Cox, L. B. (E.), 591
,, vessels, action of adrenaline, carbon dioxide and drugs on. Bouckaert, J. J., and Jourdan, F. (E.), 231
Cerebrosins, psychosin sulphate, action of, on bacteria, etc. Drury, A. N., *et al.* (E.), 129
Cerebro-spinal fluid, acetylcholine breakdown in blood and (E.), 597
,, ,, absorption of ultra-violet light by, in various disease states. Skinner, E. F. (E.), 486
,, ,, bromine content of, in nervous diseases and insanity (E.), 344
,, ,, cholesterol content of (E.), 485
,, ,, crystallization of sodium chloride in presence of. Romesco, P., Cosmulesco, I., and Serban, F. (E.), 134
,, ,, further observations on the sulphuric acid test in. Reitler, R., and Pappenheim, E. (E.), 597
,, ,, glucose content of, after withdrawal (E.), 135
,, ,, ,, ,, effect of induced hyperglycæmia on. Cohen, H., and Libman, J. (E.), 135
,, ,, glutathione of normal and pathological. Braier, B. (E.), 134
,, ,, hypertensive and hyperglycæmic action of hypertensive cerebro-spinal fluid injected into dogs. Deleonardi, S. (E.), 340
,, ,, in childhood, normal and pathological, protein fraction of. Ujsaghy, P. (E.), 135

Digitized by Google

Cerebro-spinal fluid in epidemic cerebro-spinal meningitis, sugar and glycolytic enzymes of. Hubbard, R. S., Russell, N. G. jun., and Russell, N. M. (E.), 136
,, ,, in experimental tetany, calcium content of (E.), 134
,, ,, in tuberculous meningitis, tryptophane in (E.), 134
,, ,, in vascular diseases of central nervous system. Brewer, E. D., and Hare, C. C. (E.), 136
,, ,, iso-electric point of protein. Tornu, A. (E.), 241
,, ,, of newborn infants, vitamin C content of (E.), 125
,, ,, orthophosphoric acid reaction in. Galli, G. M. (E.), 242
,, ,, origin of increased protein in, in tumours of central nervous system. Deane, J. S. (E.), 245
,, ,, phosphorus in, micro-determination of. Tropp, C., Seuberling, O., and Eckardt, B. (E.), 598
,, ,, pressure in chronic hypertension, effect of intravenous sucrose on. Murphy, F. D., Herschberg, R. A., and Katz, A. M. (E.), 237
,, ,, proteins of. Mangelschots, H. (E.), 598
,, ,, quinine test in (E.), 134
,, ,, reducing substances in. Mitolo, M. (E.), 135
,, ,, sodium chloride content of, in tuberculous meningitis. Ingham, J. (E.), 487
,, ,, simple method for determining quantity of protein and albumin-globulin ratio. Hempel, J., and Giese, L. (E.), 597
,, ,, sugar in, in animals and man. Fridman, A. P., and Arkina, R. Ch. (E.), 135
,, ,, trypanocidal activity and arsenic content of, after administration of arsenic compounds. Hawking, F., Hennelly, J. T., and Quastel, J. H. (E.), 341
,, ,, tryptophane reaction in (E.), 597
,, ,, vitamin C content of, in neurological patients (E.), 344
,, ,, ,, in (E.), 126
,, fluids of animals and man, comparative study of distribution of electrolytes in. Fridman, A. P., and Petrova, V. V. (E.), 135
Chemical composition of various divisions of nervous system. Palladin, A. V., Rashba, E. V., and Gelman, R. M. (E.), 343
,, mediation of nerve impulses, recent advances in knowledge of. Butt, H. R. (E.), 132
,, transmission of impulses through a sympathetic ganglion. Cannon, W. B., and Rosenblueth, A. (E.), 582
,, ,, of nervous influx through central synapses. Benetato, G., and Munteanu, N. (E.), 132
Child, The Growing, and its Problems. Ed. by Emanuel Miller (R.), 728
Children, mental disturbances of, art and therapy in (E.), 606
,, Reading, Writing and Speech Problems in, by Samuel T. Orton (R.), 729
Chloride content of cerebro-spinal fluid in tuberculous meningitis (E.), 487
Cholesterol, blood, and manic-depressive psychosis. Schube, P. G. (E.), 243
,, content of cerebro-spinal fluid. Holthaus, B., and Wichmann, B. (E.), 485
,, in blood, effect of emotion on. Dobreff, M., Peneff, L., and Wittkower, E. (E.), 244
Cholesterolæmia, cerebral origin of. Snachkova, A. A. (E.), 132
Choline and derivatives, biochemistry of. Kahane, E., and Levy, J. (E.), 122
,, ester formation in, and choline esterase activities of tissues *in vitro*. Quastel, J. H., Tennenbaum, M., and Wheatley, A. H. M. (E.), 487
Cholinesterase activity, comparison with hyperglycæmic index (E.) 345
,, ,, in nervous and mental disorders. Tod, H., and Jones, M. S. (E.), 342
,, in sympathetic ganglia. Brücke, F. T. v. (E.), 593
,, level in human blood-serum, effect of altering the conditions of the autonomic nervous system on the, by Maxwell Shaw Jones and Henry Tod, 202
,, oxidation of, by rat liver. Mann, P. J. G., and Quastel, J. H. (E.), 595
,, pharmacological action of three derivatives of. Wispelaere, M. de. (E.), 482
Chorea, Huntington's (E.), 482
,, minor, treatment by electro-pyrexia. Neymann, C. A., Blatt, M. L., and Osborne, S. L. (E.), 125
Circulatory changes, importance of, in mental disorder, 514
,, disorder, functional, psychological implications of, by E. Guttmann, 542
,, factor in the pathogenesis of mental disorder, by E. Krapf, 534

Digitized by Google

Circulatory reactions in psychoneuroses, studied by Schneider method (E.), 480
,, system and mental disorder: symposium at Annual Meeting, 505
CLEGG, J. L., a note on the use of diathermy in idiopathic epilepsy, 216
,, some observations on endocrines in the emotional psychoses, 52
COBB, STANLEY, A Preface to Nervous Disease (R.), 95
Compensation claim, undecided, arising from suicide of a voluntary patient, 461
,, in relation to malingering, 15
,, neurosis in relation to irritability, 64
Conditioned reflexes and psycho-analysis (E.), 334
,, ,, effect of pituitrin on (E.), 103
Confusional states in exogenous psychoses (E.), 235
Confusions, episodic, syndrome of. Kasanin, J. (E.), 481
CONRAD, K., Hereditary Disposition and Epilepsy, 99
Convolutional digitations seen in rœntgenograms of immature human skulls. Davidoff, L. M. (E.), 117
Convulsant drugs, constitution and action of. Takeuchi, S. (E.), 123
Convulsion therapy in schizophrenia, by A. Kennedy, 609
,, ,, ,, ,, by L. von Meduna (R), 727
Convulsions and syncope due to hyperactive carotid sinus reflex (E.), 236
,, brilliant vital red as anticonvulsant (E.), 603
,, during or following prolonged narcosis, 34
,, ether (E.) 123
,, experimental determination of anticonvulsant properties of some phenyl derivatives. Putnam, T. J., and Merritt, H. H. (E.), 602
,, hypoglycæmic, incidence and differential diagnosis of. Ziskind, E., Hollombe, B. S., and Bolton, R. O. (E.), 237
,, in infancy and childhood, blood sugar in (E.), 344
,, insulin, in pigeons (E.), 125
,, sugar-tolerance tests in patients with (E.), 488
,, tetanic, in a case of hysteria, 192
,, water metabolism in relation to (E.), 596
COPELAND, C. L., AND KITCHING, E. H., a case of profound dissociation of the personality, 719
,, ,, hypnosis in mental hospital practice, 316
COOK, L. C., *see* MEYER, A.
Copper content of human brain, 457
Corpus callosum, clinical diagnosis of tumours of. Cramer, F. (E.), 117
Creatine in brain in course of ontogenetic development of vertebrates. Palladin, A. V., and Rashba, H. (E.), 230
Creatinuria in post-encephalitic parkinsonism. Porta, V., and Pelliecoli, V. G. (E.), 241
,, spontaneous, in nervous diseases. Kostakow, S. (E.), 133
Criminal types as seen by the psycho-analyst. Alexander, F., and Saul, L. J. (E.), 479
Criminology epitomes, 346
Cryptorchidism associated with behaviour problems. Molitch, M. (E.), 587
Curarine and acetylcholine, antagonism between. Briscoe, G. (E.), 123
CURRAN, DESMOND, the differentiation of neuroses and manic-depressive psychoses, 156
Cysticercosis of brain. Stern, I. B., and Baskins, N. A. (E.), 110

DAVIS, J. H., Principles and Practice of Recreational Therapy for the Mentally Ill (R.), 574
DAX, E. CUNNINGHAM, AND PENTREATH, E. V. H., mental observation wards, 347
Delinquency, aspects of temperament in, 268
Delirious episodes associated with artificial fever (Kettering hypertherm) (E.), 483
Delirium, acute, intravenous saline and prolonged narcosis in, 27
,, tremens, comprehensive management of. Piper, P., and Cohn, J. V. (E.), 239
Dendrobine, detoxication of, by sodium amytal. Chen, K. K., and Rose, C. L. (E.), 120.
Depression, construction of (psycho-analytic). Gerö, G. (E.), 236
,, neurotic and manic-depressive, criticism of criteria, 160
Diabetes insipidus. Salmon, A. (E.), 242
Diabetic acidosis, cerebral lesions in (E.), 109
Diathermy, a note on the use of, in idiopathic epilepsy, by J. L. Clegg, 216
Dichlorodifluoromethane, action of, on nervous system. Brenner, C. (E.), 340
DIDE, M., L'Hystérie et l'Evolution Humaine (R.), 578
Dielectric behaviour of organic Zwitter ions. Hausser, I. (E.), 130
DIETHELM, OSCAR, Treatment in Psychiatry (R.), 573
DILLON, FREDERICK, and MASANI, K. R., Psychosis or malingering ?, 15

Digitized by Google

Diphtheria, neurological, symptomatology in. Salkan, D. M., and Epstein, P. D. (E.), 590
Disseminate sclerosis, connective-tissue reaction in. King, L. S. (E.), 242
,, ,, sensory and other aspects of. Brickner, R. M. (E.), 117
Divorce for "incurable" insanity, views on, 500
Dreams and dream interpretation of California Indians. Toffelmier, G., and Luomala, K. (E.), 106
Drury, K. K., and Freer, C. E. J., an undecided compensation claim arising from the suicide of a voluntary patient, 461
Dysentery, control of, in mental hospitals, 298
,, endemic bacillary, by J. J. B. Martin, 289
,, factors facilitating conveyance, 296
,, incidence of, 289
,, organisms causing, 292
,, use of vaccines in, 306

Eager, R., Hints to Probationer Nurses in Mental Hospitals (R.), 330
Ego-development and certain character problems. Hendrick, I. (E.), 232
Electro-encephalogram in epilepsy, the, by F. L. Golla, S. Graham and W. Grey Walter, 137
Electro-encephalography: action of mescaline on α-waves (E.), 602
Electropyrexia in treatment of general paralysis (E.), 483
,, ,, chorea minor (E.), 125
,, ,, idiopathic epilepsy, 216
,, ,, syphilis (E.), 483
,, ,, ,, and neurosyphilis (E.), 607
Emotion, effect of, on blood cholesterol (E.), 244
Emotional expression: associated facial, vocal and respiratory components of. Magoun, H. W., *et al.* (E.), 478
,, ,, role of mid-brain and pons in (E.), 478
Encephalitic, post-, parkinsonianism, pathology of (E.), 598
,, ,, use of benzedrine in (E.), 484
Encephalitis, epidemic, and teaching problems. Bortnik, N. V. (E.), 108
,, post-, atropine in cure of (E.), 240
,, ,, creatinuria in (E.), 241
Encephalographic sign, pathognomonic, of subdural hæmatoma. Dyke, C. G. (E.), 116
Encephalography, effect of, on blood-sugar level of children. Bradley, C. (E.), 241, 593
,, with anæsthetic gases. Aird, R. B. (E.), 590
,, with ethylene. Newman, H. (E.), 237
Endocrine disorder, familial psychoses associated with, by T. A. Munro, 707
,, disorders, in states of excitation. Turques, I. (E.), 590
,, glands from point of view of neuro-psychiatry. De Loverdo, G. (E.), 481
Endocrinopathies among young delinquents, statistical survey of. Taylor, N. M., and Schaeffer, R. L. (E.), 346
Endocrines in the emotional psychoses, some observations on, by J. L. Clegg, 52
Enzymes in brain: effect of dichlorodiethylsulphone on brain respiration. Peters, R. A. (E.), 133
,, ,, glycerophosphatase. Cedrangolo, F. (E.), 129
,, ,, glycolysis and phosphate exchange. (E.), 131
Ephedrine as antidote for acute barbiturate poisoning. Abbatecola, B. (E.), 600
Epilepsy and Heredity (R.), 99
,, and hysteria, 691
,, electro-encephalogram in, by F. L. Golla, S. Graham, and W. Guy Walter, 137
,, essential, 687
,, ,, and symptomatic, differences exhibited by electro-encephalogram, 145
,, ,, ,, ,, by studies on twins, 100
,, experimental: change in protective qualities of serum. Nekludov, V. (E.), 130
,, fatal case of *status epilepticus*, 568
,, due to cysticercosis (E.), 110
,, idiopathic, a note on the use of diathermy in, by J. L. Clegg, 216
,, in mental defectives with congenital syphilis, treated by acetarsol, 382
,, symptomatic, with hysteroid trait on a background of chronic sepsis. Skvortzow, K. A., and Skipetrov, A. I. (E.), 107
,, traumatic, acetylcholine in (E.), 131
,, treatment of, with snake venom (E.), 237
,, treatment with calcium bromide. Moore, M. T. (E.), 124
,, with psychosis, the treatment of, with prominal, by D. E. Sands, 703

Digitized by Google

Epileptic abreaction, 690
,, deterioration, progressive, 683
,, reactions: an attempt at classification, by Arthur Guirdham, 679
,, retardation, 684
Erythrocyanosis crurum puellaris, 428
Eserine and related compounds, and acetylcholine, action of, on central nervous system. Schweitzer, A., and Wright, Samson (E.), 604
Ether convulsions. Hoseason, E. S. (E.), 123
Evolution and the nervous system, 3
Exhibitionism and exhibitionists. Christoffel, H. (E.), 106
Exogenous psychoses. Rafkin, I. G. (E.), 235
Exophthalmic goitre, action of mid-brain narcosis on blood iodine in. Fenz, E., and Uiberrak, K. (E.), 606
,, ,, and psychosis. Bram, I. (E.), 589
,, ,, complicated by cranial nerve palsy. Bogorad, F. A., and Retchnik, M. A. (E.), 110

Familial psychosis associated with endocrine disorder, by T. A. Munro, 707
Family size in relation to "neuropathic" abnormalities, 249
Fatigue, in relation to irritability, 63
Fat metabolism, cerebral control of. Molnar, I., and Marsovszky, P. (E.), 105
,, ,, in epidemics, central regulation of. Perutz, A., *et al.* (E.), 130
FRASER, RUSSELL, a case of hysteria showing spontaneous hyperventilation tetany, 190
FREER, C. E. J., *see* DRURY, K. K.
Freud and Marx, by R. Osborn (R.), 580
FREUD, SIGMUND, Inhibitions, Symptoms and Anxiety (R.), 222
Friendship-Love in Adolescence, by N. M. Jovetz-Tereshchenko (R.), 223

"G," deterioration of, in psychotic patients (E.), 235
General paralysis, genealogical studies in. Selzer, H. (E.), 335
,, ,, malaria treatment of (E.), 124
,, ,, presence of spirochæte in vegetative nervous system in. Benvenuti, M. (E.), 127
,, ,, treated with mosquito-inoculated malaria. Kusch, E., Milam, D. F., and Stratman-Thomas, W. K. (E.), 482
GOLLA, F. L., GRAHAM, S., and WALTER, W. GREY, the electro-encephalogram in epilepsy, 137
,, some remarks on the physiology of the cerebral circulation, 505
Gliosis, diffuse white matter, in mental defectives, by A. Meyer and L. C. Cook, 258
Glycerophosphoric acid and brain metabolism (E.), 487
Glycolysis and phosphate exchange in cell-free brain extracts. Euler, H., *et al.* (E.), 131
GRAHAM, S., *see* GOLLA, F. L.
GUILFORD, J. P., Psychometric Methods (R.), 228
GUIRDHAM, ARTHUR, epileptic reactions: an attempt at classification, 679
GURWITSCH, A. G., Mitogenetic Analysis of the Excitation of the Nervous System (R.), 577
GUTTMANN, E., and MAYER-GROSS, W., schema for the examination of organic cases, 440
,, psychological implications of functional circulatory disorder, 542

Hæmatoma, subdural, encephalographic sign of. Dyke, C. G. (E.), 116
HARDING, ROSAMOND E. M., Towards a Law of Constructive Thought (R.), 575
Headache, ætiology of. Brewer, E. P. (E.), 481
,, after ventriculography (E.), 481
,, post-traumatic and subdural traction (E.), 110
Headaches, post lumbar-puncture, clinical studies in. Heldt, T. J., and Whitehead, W. S. (E.), 483
Head injury in boxing, relation to vascular disturbances, 545
,, ,, psychosis or malingering following, 15
Hearing in patients with intracranial tumours (E.), 112
Heart disease, functional and organic, psychological symptoms in, 548
,, failure, psychoses associated with, 551
Hemiplegia, trophic disturbances in. Marinescu, G., *et al.* (E.), 337
HENRY, G. H., Essentials of Psychopathology (R.), 96
Hereditary Disposition and Epilepsy, by K. Conrad (R.), 99

Digitized by Google

Heredity in neurosis, observations on. McInnes, R. G. (E.), 337
,, psychopathic, with special reference to manic-depressive psychosis. Ormea, A. (E.), 336
,, studies in general paralysis (E.), 335
,, study: incidence of "neuropathic" conditions in the relatives of normal persons, 247
Hints to Probationer Nurses in Mental Hospitals, by R. Eager (R.), 330
Histamine, action of, on cerebral blood-vessels. Bruch, H. (E.), 606
Homosexuality, unconscious, and Tolstoy's "Kreutzer Sonata" (E.), 334
HONAN, M. S., *see* MOLONY, C. B.
HOPKINS, F., attempted suicide: an investigation, 71
Hormone-growth, effect of, on brain and brain-weight–body-weight relations. Rubinstein, H. S. (E.), 230
HORSLEY, J. S., therapeutic narcosis with soneryl, 25
Huntington's chorea: its incidence in the Scottish Highlands. McWilliam, W. (E.), 482
Hydrocephalus, from obstruction of aqueduct of Sylvius (E.), 114
Hypertension, arterio-sclerosis and heart failure, psychoses associated with, W. Mayer-Gross, 551
,, chronic, effect of intravenous sucrose on (E.), 237
,, psychiatric characteristics of, 536
Hypertensive cerebro-spinal fluid injected into dogs, effect of, 340
Hyperthyroidism, experimental, phospholipides in brain, etc., in (E.), 485
Hypnosis and the vegetative nervous system. Bogolepov, N. K. (E.), 107
,, in a case of dissociation of personality, 719
,, in mental hospital practice, by C. L. Copeland and E. H. Kitching, 316
Hypoglycæmia, cerebral lesions in. Baker, A. B., and Lufkin, N. H. (E.), 243
Hypoglycæmic convulsions (E.), 237
,, shock treatment, *see* Insulin.
Hypogonadism in schizophrenics, 58
Hypotension, postural. Ellis, L. B., and Haynes, F. W. (E.), 110
Hypothalamus and cardio-vascular reactions. Van Bogaert, A. (E.), 104
,, in nervous regulation of pituitary function (E.), 582
,, lesions of, effect of, on carbohydrate metabolism. Cleveland, D., and Davis, L. (E.), 582
,, stimulation of, inducing liberation of adrenaline and sympathin (E.), 584
Hysteria, a case of, showing spontaneous hyperventilation tetany, by Russell Fraser, 190
,, and schizophrenia, relation between. Claude, H. (E.), 587
Hysterical dissociation of personality, case of, 719
,, reactions, relation to circulatory disturbances, 543
Hystérie et Évolution Humaine, by M. Dide (R.), 578

Inhibitions, Symptoms and Anxiety, by Sigmund Freud (R.), 222
,, symptoms and anxiety. Freud, S. (E.), 105, 233
Iron, copper and manganese content of the human brain, the, by A. H. Tingey, 452
Irritability as a symptom in manic-depressives, by W. Mayer-Gross, 61
,, in toxic and fatigue states, 63, 64
Intellectual Functions of the Frontal Lobes, by R. M. Brickner (R.), 575
Intelligence, investigation of deterioration of general ("G"), in psychotic patients. Harbinson, M. R. (E.), 235
Intermetamorphosis, delusion of. Daumezon, G. (E.), 336
Introjection, on. Fuchs, S. H. (E.), 586
Iodine, in blood, in exophthalmic goitre, action of mid-brain narcosis on (E.), 606
Insane, some observations on the care of the (Presidential Address), by Douglas McRae, 489
Institutional life, reaction to, of various constitutional types, 283
Insulin convulsions in pigeons. Moracci, E. (E.), 131
,, hypoglycæmia, mechanism of convulsions in. Drabkin, D. L., and Ravdin, I. S. (E.), 339
,, response in acromegaly. Berg, B. N. (E.), 591
,, shock therapy, an unusual complication of, by C. B. Molony and M. S. Honan, 630
,, ,, Treatment in Schizophrenia, A Study of, by Isabel G. H. Wilson (R.), 218

JONES, MAXWELL SHAW, and TOD, HENRY, the effect of altering the conditions of the autonomic nervous system on the choline esterase level in human blood serum, 202
JOVETZ-TERESHCHENKO, N. M., Friendship-Love in Adolescence (R.), 223

Digitized by Google

KITCHING, E. H., and COPELAND, C. L., hypnosis in mental hospital practice, 316
,, and DIGNAM, A., the excretion of sodium barbitone in patients suffering from mental illness, 40
KENNEDY, A., convulsion therapy in schizophrenia, 610
KIMBER, W. J. T., Practical Psychology for Nurses and Other Workers in Mental Hospitals (R.), 330
KRAPF, E., the circulatory factor in the pathogenesis of mental disorder, 534

LANGDON-BROWN, Sir W., the biology of social life (Maudsley Lecture), 1
Law of Creative Thought, Towards a, by Rosamond E. M. Harding (R.), 575
Lead and arsenic, some neurological signs produced by. Cornwall, L. H. (E.), 116
Lecithin and cephalin, antigenic properties of. Wadsworth, A., Maltaner, E., and Maltaner, F. (E.), 133
Lecithins and cephalins of brain, evidence of selection in building-up of. McConnell, K. P., and Sinclair, R. G. (E.), 243, 596
LECOMTE DU NOÜY, Biological Time (R.), 97
Left-handedness in prehistoric peoples. Peola, P. (E.), 229
LEWIS, NOLAN D. C., Research in Dementia Præcox (R.), 573
Lipides, brain, and learning ability of albino rats. Alm, O. W., and Whitnah, C. H. (E.), 343
,, ,, selection in building-up of (E.), 243
,, metabolism of, action of nervous system on. Cedrangolo, F. (E.), 134
Lumbar puncture, headache following (E.), 483
LUNDHOLM, H., The Psychology of Belief (R.), 578

MCRAE, DOUGLAS, some observations on the care of the insane (Presidential Address), 489
Magnesium salts, use of, in psychiatry. Morsa, G. (E.), 242
Malarial psychoses, contribution to the study of. Sklar, N. I., 588
Malaria therapy in general paralysis, alkali reserve before and after. Paulian, D., and Tanasesco, G. (E.), 124
,, ,, of neurosyphilis, delayed results of. Tarsis, F. I., and Landesman, J. U. (E.), 124
,, treatment of general paralysis. Barnacle, C. H., Ebaugh, F. G., and Ewalt, J. R. (E.), 124
,, ,, ,, ,, (mosquito inoculated) (E.), 482
,, ,, ,, ,, views on, 499
Malingering, psychosis or, by F. Dillon and K. R. Masani, 15
Manganese content of human brain, 458
Maniacal states, endocrine disorders in (E.), 590
Manic-depressive insanity, glucose tolerance in (E.), 132
,, psychoses and neuroses, differentiation of, by Desmond Curran, 156
,, ,, blood-platelet count in, 182
,, ,, blood bromine in (E.), 128
,, ,, exogenous ætiological factors in. Dickmeiss, P. (E.), 235
,, psychoses and blood cholesterol (E.), 243
,, ,, chronic forms of. Halberstadt, G. (E.), 337
,, ,, heredity in (E.), 336
,, ,, endocrines in, 56
,, ,, observations on disturbances in gastric secretion in. (Hedström, U. (E.), 594
,, states, periodic syndromes exclusive of (E.), 481
,, symptoms in identical twins. Selzer, H. (E.), 336
Manic-depressives, irritability as a symptom in, by W. Mayer-Gross, 61
Manual reflex. Pool, J. L. (E.), 591
MARTIN, J. J. B., endemic bacillary dysentery, 289
MASANI, K. R., and DILLON, F., psychosis or malingering ?, 15
Maudsley Lecture, the seventeenth: the biology of social life, by Sir W. Langdon-Brown, 1
MAYER-GROSS, W., irritability as a symptom in manic-depressives, 61
,, psychoses associated with hypertension, arterio-sclerosis and heart failure, 551
,, and GUTTMANN, E., schema for the examination of organic cases, 440
MEDUNA, L. VON, Convulsion Therapy in Schizophrenia (R.), 727
Melancholia, involutional, treated by theelin. Werner, A. A., *et al.* (E.), 118
Meninges, permeability of. de Arric. (E.), 597

Digitized by Google

Meningitis, cerebro-spinal, sugars and glycolytic enzymes of cerebro-spinal fluid in (E.), 136
,, tuberculous, pathology of. Moritz, Denes v., and Kulcsar, M. (E.), 128
,, ,, tryptophane in cerebro-spinal fluid in (E.), 134
,, ,, ,, reaction in cerebro-spinal fluid in diagnosis of (E.), 597
Mental defectives, congenital syphilis amongst, treatment by acetarsol, 372
,, ,, diffuse white matter gliosis in, by A. Meyer and L. C. Cook, 258
,, Nursing (Simplified), by O. P. Napier Pearn (R.), 330
,, Treatment Act, discussion on, in relation to the suicide of a voluntary patient, 466
,, ,, ,, in relation to mental observation wards, 359
,, ,, ,, "temporary" patients under, analysis of 30 cases, 367
Mescaline, action of, on α-waves (Berger rhythm). Chweitzer, A., Geblewitz, E., and Libersch, W. (E.), 602
,, toxicology and pharmacology of. De Nito, G. (E.), 604
Methylimidazole, circulation under action of. Axmacher, F. (E.), 605
,, stimulating and restorative properties of. Axmacher, F. (E.), 605
Meyer, A., the histopathological criteria of vascular disturbances in the brain. (Abstract), 509
,, and Cook, L. C., diffuse white matter gliosis in mental defectives, 258
Microcephaly, white matter gliosis in, 259
Mid-brain, importance of, in water metabolism. Pette, H. (E.), 345
Migraine: a critique of the hypophyseal theory. Timme, W. (E.), 113
,, mental disorder accompanying, 544
,, menstrual, treatment of. Moffat, W. M. (E.), 239
Miller, Emanuel (ed. by), The Growing Child and its Problems (R.), 728
Minski, Louis, a note on some vasomotor disturbances in schizophrenia, 437
,, Psychiatry for Nurses (R.), 330
,, and Wootton, L. H., "temporary" treatment: analysis of 30 cases, 366
Mitogenetic Analysis of the Excitation of the Nervous System, by A. G. Gurwitsch (R.), 577
Molony, C. B., and Honan, M. S., an unusual complication of insulin shock therapy, 630
Mongolism, white-matter gliosis in, 261
Morphinism, dispensary observations on. Kantorovitch, N. V. (E.), 107
,, nerve-centre system in, study of. Hayashi, A. (E.), 602
Movement, variation of precision of, with age during adolescence. Sparrow, W. J. (E.), 585
Munro, T. A., familial psychoses associated with endocrine disorder, 707
Murderer, The Unknown, by Theodore Reik (R.), 581
Muscular dystrophies, influence of follicular hormone on creatine metabolism in. Paleari, A. (E.), 346
Myoclonus, rhythmic, of muscles of palate, larynx, etc. Davison, C., Riley, H. A., and Brock, S. (E.), 116

Narcolepsy. Speirs, G. O., and Speirs, R. E. (E.), 338
,, mechanism of. Lion, E. G. (E.), 237
,, treatment of, with benzedrine (E.), 599
Narcosis and dehydrogenation. Galli, G., and Radici, G. (E.), 121
,, and glutathione. Galli, G., and Mingazzini, I. (E.), 120
,, and respiratory centre (E.), 240
,, continuous, value of, in the treatment of mental disorder, by H. A. Palmer, 636
,, mid-brain, action of, on blood iodine, in exophthalmic goitre (E.), 606
,, prolonged, analysis of results of, 652
,, ,, body habitus and results in, 657
,, ,, catatonia induced by, 32
,, ,, complications of, 648
,, ,, cravings in, 32
,, ,, criticism of insulin-glucose method, 663
,, ,, discussion of mode of action, 665
,, ,, duration of, 31
,, ,, dynamics of schizophrenia under the influence of. Gilarovski, V. A. (E.), 119
,, ,, epilepsy during or following, 34
,, ,, induced by sodium amytal, abridged method. Broder, S. B. (E.), 342
,, ,, technique of, 644
,, ,, therapeutic results of, 38, 652

Digitized by Google

Narcosis, prolonged, use of intravenous saline in, 27
,, ,, with Cloetta's mixture, technique of. Richter, G. E., and Stepanov, P. N. (E.), 604
,, therapeutic, with soneryl, by J. S. Horsley, 25
Narcotics, action of, on autonomic nervous system. Moraeus, B. (E.), 338
,, comparative actions of various. Brusilovskaya, A. I., and Lazarev, N. V. (E.), 120
,, disturbance of glucose-tolerance caused by (E.), 597
,, effect of antipyretics on action of. Girndt, O., and Huhn, O. (E.), 605
,, ,, on tissue oxidations. Jowett, M., and Quastel, J. H. (E.), 341
,, effect of cardiazol and ephedrine on animals treated with. Hofmann, M. (E.), 605
,, in animal experimentation. Fromherz, K. (E.), 340
,, *see also under* Barbiturates, etc.
Nerve proteins, unimolecular films of. Fourt, L., and Schmitt, F. O. (E.), 343
,, substance, presence and distribution of reducing substance in. Mitolo, M. (E.), 104
Nervous Disease, A Preface to, by Stanley Cobb (R.), 95
,, System, Excitation of, Mitogenetic Analysis of (R.), 577
,, ,, of *Bdellonia candida*, observations on. Elwyn, A. (E.), 115
,, ,, peripheral, pathological changes of, under action of electrical currents. Zolotova, K. A., and Zinkin, A. M. (E.), 583
,, ,, variations in weight, water content and potassium from birth to maturity. Leulier, A., and Bernard, A. (E.), 583
Neuroglia, proliferation of, in mental deficiency, 258
Neurology epitomes, 109, 263, 337, 481, 590
Neuro-muscular junction: antagonism between calcium and curarizing agencies. Feng, T. P. (E.), 333
,, contracture in eserinized muscle. Feng, T. P., and Shen, S. C. (E.), 333
"Neuropathic" conditions in the relatives of normal persons, an inquiry into the incidence of; report by Mental Deficiency Committee, 247
,, patients in sanatoria for nervous cases. Emdina, S., Voronova, M., and Asarkh, M. (E.), 108
Neuroses and manic-depressive psychoses, the differentiation of, by Desmond Curran, 156
Neurosyphilis, treatment of, by electropyrexia (E.), 607
,, *see also under* Syphilis
Neurotics, vasomotor lability in, 547
Nicotine, action of, on nerve excitability, a new interpretation of Langley's phenomenon. Chauchard, P. (E.), 602
Niemann-Pick's disease, brain sphingomyelin in. Tropp, C., and Eckhardt, B. (E.), 593
Nitrogen in blood, neuro-central regulation of. Jedlowski, P. (E.), 131
Nystagmic head deviation: selective action of an aliphatic bromine compound. Loewe, S. (E.), 603

Observation wards, mental, a discussion of their work and its objects, by E. U. H. Pentreath and E. Cunningham Dax, 347
,, ,, cases of attempted suicide in, 71
,, ,, mental, disposal of cases, 358
,, ,, ,, types of patients admitted to, 355
Operative treatment of certain psychoses. Moniz, E. (E.), 118
Optic nerve, visual disturbances produced by focal intracranial lesions implicating. McKendree, C. A., and Doshay, L. J. (E.), 114
Organic cases, schema for the examination of, by W. Mayer-Gross and E. Guttmann, 440
Orientation in spatial direction, disorders of. Vikker, J. L. (E.), 109
Osborn, R., Freud and Marx (R.), 580
Orton, Samuel T., Reading, Writing and Speech Problems in Children (R.), 729
Ovarian follicular hormone (theelin) in treatment of melancholia (E.), 118

Padded rooms, use of, 497
Paddle, K. C. L., acetarsol in the treatment of late congenital syphilis amongst mental defectives, 372
Palmer, H. A., the value of continuous narcosis in the treatment of mental disorder, 636
Paralyses, psychogenic, curve pattern of muscular contraction and tonus mobility in. Shirokov, A. A. (E.), 589

Digitized by Google

Parkinsonianism, pathology of. Neustaedter, M., and Liber, A. F. (E.), 598
,, *see also under* Encephalitis
Patellar reflex, action of adrenaline on (E.), 339
,, ,, effect on, of stimulation of vagus, etc. Schweitzer, A., and Wright, S. (E.), 333
,, ,, functional changes in, in the psychoses. Strecker, E. A., and Hughes, J. (E.), 480
Pathology and Biochemistry epitomes, 125, 241, 342, 485, 591
PEARN, O. P. NAPIER, Mental Nursing (Simplified) (R.), 330
Perceptual tests, new series of. Penrose, L. S., and Raven, J. C. (E.), 232
Periodic nervous and mental syndromes, exclusive of manic-depressive states. Baruk, M. H., and Gévaudan, Mle. (E.), 481
Permeability of nerve centres. De Marco, R. (E.), 231
,, ,, ,, effects of tetanus toxin on (E.), 339
,, ,, ,, morphine and venoms on. Marco, R. de (E.), 342
PENTREATH, E. U. H., and DAX, E. CUNNINGHAM, mental observation wards, 347
Personality, a case of profound dissociation of the, by C. L. Copeland and E. H. Kitching, 719
,, psychopathic, irritability in, 65
PETERSEN, WILLIAM F., The Patient and the Weather (R.), 227
Pharmacology and Treatment epitomes, 118, 237, 338, 482, 599
Phenobarbital poisoning. Taylor, H. W., and Schram, M. (E.), 338
Phosphatide-splitting property of serum from normal and psychotic individuals. Freund, E., Lustig, B., and Heimann, M. (E.), 485
Phospholipides of brain, kidneys and heart of white rats in experimental hyperthyroidism. Weil, A. (E.), 485
Phosphorus compounds in brains of various animals, amount of. Boldyreva, N. V. (E.), 592
,, content of brain, effect of age on. Epelbaum, S. E., *et al.* (E.), 343
Phrenosins and kerasin, fatty acids of. Chibnall, A. C., Piper S. H., and Williams, E. F. (E.), 486
Physostigmine, action of, on autonomic ganglia. Koppanyi, T., Dille, J. M., and Linegar, C. R. (E.), 240
PICKWORTH, F. A., cerebral ischæmia and mental disorder, 512
Pituitrin, effect of, on conditioned reflexes. Asratyan, E. (E.), 103
Picrotoxin, effects of hypnotics on, contribution to the problem of the cold centre. Rosenthal, F., and Wallach, G. (E.), 120
Pilocarpine, effect of, on neurogenic urinary retention. Dean, J. S. (E.), 483
Pituitary adenoma, visual studies in. Franklin, C. R. (E.), 112
,, anterior, relation to menstrual migraine (E.), 239
,, deficiency in manic-depressive psychoses, 60
,, function, nervous regulation of. Roussy, G., and Mosinger, M. (E.), 582
,, human, development and constituents of. Tilney, F. (E.), 113
,, in relation to migraine, theories of (E.), 113
Poliomyelitis, anterior, and ascaris infestation (E.), 337
,, in the alcohol addict, ætiology of. Jolliffe, N., and Colbert, C. N. (E.), 111
Polypeptidæmia, contribution to study of. Artur, Boucher and Coulonjou (E.), 245
Potassium and calcium ions, changes in concentration of, in perfusates. Gavrilescu, N., and Ionescu, N. (E.), 592
,, in brain in vitamin B_1 deficiency. Kinnersley, H. W. (E.), 126
,, in central nervous system, variations in (E.), 583
Pregnancy, biochemistry of nervous system during. Rashba, O. (E.), 131
PRENTICE, DAVID, syphilis in mental hospital practice, 472
Presenile psychoses, relation to vasomotor disturbance, 557
Presidential Address, by Dr. Douglas McRae, 489
Prison routine and discipline, effect of, on some types of abnormal delinquents, 283
Prognosis in the Neuroses, An Enquiry into, by T. A. Ross (R.), 222
Projection, on. Feigenbaum, D. (E.), 233
Prominal, treatment of epilepsy with psychosis by, by D. E. Sands, 703
Protagon fraction of brain, new hapten in. Schwab, E. (E.), 129
Psychasthenia, on the nature of. Ozeretzkovski, D. S. (E.), 108
Psychiatry epitomes, 107, 235, 335, 480, 587
Psychiatry for Nurses, by Louis Minski (R.), 330
,, Theory and Practice of, by William S. Sadler (R.), 225
Psychoanalysis and conditioned reflexes. Schilder, P. (E.), 334
,, and criminal types (E.), 479
,, inhibitions, symptoms and anxiety. Freud, S. (E.), 105

Digitized by Google

Psychoanalysis, medical value of (addenda). Alexander, F. (E.), 334
,, psychological factors in urological disease. Menninger, K. (E.), 335
,, some anthropological perspectives on. du Bois, Cora (E.), 586
,, therapeutic results of, symposium on the theory of. Glover, E., *et al.* (E.), 585
Psychology and psychopathology epitomes, 105, 232, 334, 479, 585
Psychology, Practical, for Nurses and Other Workers in Mental Hospitals, by W. J. T. Kimber (R.), 330
Psychometric Methods, by J. P. Guilford (R.), 228
Psychopathology, Essentials of, by G. H. Henry (R.), 96
Psychoneuroses and manic-depressive psychoses, differentiation of, 156
,, and neuroses. Yarkin, J. C. (E.), 479
,, neuro-circulatory reactions in, studied by the Schneider method. McFarland, R. A., and Huddleson, J. H. (E.), 480
Psychoneurotics in mental hospitals, treatment of, 316
Pyretotherapy, for chorea minor (E.), 125
,, of schizophrenia by sulphur (E.), 607
,, for general paralysis (E.), 483
,, sulphur, in schizophrenia (E.), 118
,, sympathetic factors in. Alagia, D. P., and Flannery, D. L. (E.), 341
,, treatment of tabes dorsalis by Kettering hypertherm (E.), 125
,, *see also under* Electropyrexia

Quinine test, a new cerebro-spinal reaction. Takata, M. (E.), 134

Raynaud's disease, differentiation from acrocyanosis, 430
Reality and the unconscious. French, T. M. (E.), 479
Recreational Therapy for the Mentally Ill, Principles and Practice of, by J. H. Davis (R.), 574
Reducing substance of brain, nature of. Bonsignore, A., and Ardy, C. (E.), 126
Reik, Theodor, The Unknown Murderer (R.), 581
Renal efficiency in relation to barbitone excretion, 49
,, function tests in psychoses, 208
Research in Dementia Præcox, by Nolan D. C. Lewis (R.), 573
Respiration of brain, oxidation-reduction potentials in strychnine and cocaine poisoning. Fomin, S. V., and Strashesko, D. N. (E.), 133
Respiratory centre and narcosis. Mansfeld, G., and Tyukody, F. v. (E.), 240
Ross, T. A., An Enquiry into Prognosis in the Neuroses (R.), 222

Sadler, William S., Theory and Practice of Psychiatry (R.), 225
Sands, D. E., the treatment of epilepsy with psychosis by prominal, 703
Schema for the examination of organic cases, by W. Mayer-Gross and E. Guttmann, 440
Schizophasia, a note on some vasomotor disturbances in, by Louis Minski, 437
Schizophrenia, acrocyanosis in, 413
,, analysis of a case of. Sjöden, B. (E.), 587
,, and hysteria, relationship between (E.), 587
,, A Study of Hypoglycæmic Shock Treatment in, by Isabel G. H. Wilson (R.), 218
,, convulsion therapy in, 610, 727
,, dynamics of, under prolonged narcosis (E.), 119
,, endocrines in, 56
,, familial psychosis resembling, associated with endocrine disorder, 707
,, fever treatment of, with sulphur in oil. Shapiro, L. B., and Read, C. F. (E.), 607
,, glutathione content of blood in. Ljungberg, E. (E.), 594
,, certain hormonal disorders in. Turkevitch, O. M., and Gershanovitch, Z. S. (E.), 588
,, interrelations of, and heterogenic organic diseases of central nervous system. Grinberg, M. D. (E.), 588
,, Research in (R.), 573
,, treatment of, with tissue hystolysates of Tushnov. Maiorov, F. P., *et al.* (E.), 118
,, value of sulphur pyretotherapy in. Imber, I. (E.), 118
Science, The Logical Structure of, by A. Cornelius Benjamin (R.), 575
Sclerosis, diffuse and multiple, connective-tissue reaction in. King, L. S. (E.), 242
Scopophilic instinct and identification. Fenichel, O. (E.), 480
Self in Psychology, The, by A. H. B. Allen (R.), 575
Senile dementia without cerebral atheroma. Marchand, L. (E.), 589

Digitized by Google

Sepsis, chronic, as background to symptomatic epilepsy (E.), 107
Sleep, changes in serum calcium and protein during (E.), 132
,, in relation to irritability, 63
,, pathological, in relation to tumours of base of brain. Cox, L. B. (E.), 591
Snake venom (moccasin) in treatment of epilepsy. Finkelman, I. (E.), 237
Social life, biology of, by Sir W. Langdon-Brown, 1
Soneryl, therapeutic narcosis with, by J. S. Horsley, 25
Speech problems in children, 729
Spinal reflexes, action of stimulant on. Knoefel, P. K., and Murrell, F. C. (E.), 123
Spiritual Healing, The Background of, by A. Graham Ikin (R.), 580
Status epilepticus, a fatal case of, with post-mortem findings, by A. M. Wyllie, 568
STERN, E. S., acrocyanosis, 408
Stimulant-depressant action of caffeine, etc., analysis of. Maloney, A. H. (E.), 123
Strychnine and barbiturate, urine of animals after separate injection of (E.), 119
,, poisoning, oxidation-reduction potentials of brain in (E.), 133
Subdural traction and post-traumatic headache. Penfield, W., and Norcross, N. C. (E.), 110
Sugar in blood, comparison of hyperglycæmic index and cholinesterase activity. Tod, H., and Jones, M. S. (E.), 345
,, ,, disturbance of glucose-tolerance test caused by hypnotics in clinical doses. Tod, H. (E.), 597
,, ,, effect of acetylcholine, etc., on (E.), 244
,, ,, ,, encephalography on (E.), 593
,, ,, ,, ,, on level of (E.), 241
,, ,, ,, environmental temperature on, in heat-punctured rabbit. Ichikawa, K. (E.), 104
,, ,, ,, injury of cerebellum on (E.), 103
,, ,, ,, lesions of hypothalamus on (E.), 582
,, ,, in childhood, cerebral regulation of. Roboz, P., and Rausnitz, E. (E.), 344
,, ,, in convulsions of infancy and childhood. Maclean, Margaret B. (E.), 344
,, ,, increased, effect on glucose content of cerebro-spinal fluid (E.), 135
,, intravenous, in cases of chronic hypertension (E.), 237
,, metabolism in mental disorders, glucose tolerance in manic-depressive insanity. Tod, H. (E.), 132
Sugar-tolerance tests in two hundred patients with convulsions. Dreary, H. H. (E.), 488
Suicide and mental disease. Jamieson, G. R. (E.), 107
,, attempted: an investigation, by F. Hopkins, 71
,, ,, causes of, 84
,, of a voluntary patient, an undecided compensation claim arising from, by K. K. Drury and C. E. J. Freer, 461
Suicide, on, by R. Weichbrodt (R.), 730
,, statistics of, 72
Sylvius, aqueduct of, obstruction of, by neoplastic and non-neoplastic processes. Stookey, B., and Scarff, J. (E.), 114
Sympathetic ganglion, acetylcholine mechanism of (E.), 245
Sympathin and adrenaline, liberation of, by stimulation of hypothalamus (E.), 584
,, in chemical mediation of nerve impulses (E.), 132
Syncope and convulsions due to hyperactive carotid sinus reflex. Weiss, S., *et al.* (E.), 236
Syphilis and neurosyphilis treated by electropyrexia. Prior, G. P. V. (E.), 607
,, congenital, amongst mental defectives, treatment by acetarsol, 372
,, ,, treated by acetarsol, serological reactions in, 376
,, effect of artificial fever on. Neymann, C. A. (E.), 483
,, in mental hospital practice, by David Prentice (with discussion), 472

Tabes dorsalis, pyretotherapy in. Bennett, A. H. (E.), 125
Temperament in adolescent male offenders, aspects of, by H. T. P. Young, 268
Temperature regulation, hypnotics and picrotoxin (E.), 120
,, ,, role of central nervous system and thyroid gland in. Inekutz, B., jun. (E.), 583
"Temporary" treatment, an analysis of 30 cases, by L. H. Wootton and Louis Minski, 366
Tetanus toxin, effect of, on permeability of nerve-centres. Isanto, A. D. (E.), 339
Tetany, convulsions during or following prolonged narcosis, 34
,, experimental, calcium content of cerebro-spinal fluid in. (E.), 134
,, ,, water, calcium and potassium of brain in. Parhon, C. I., and Cahane, M. (E.), 345

Digitized by Google

Tetany, spontaneous hyperventilation, in a case of hysteria, 190
,, ,, overbreathing, role of calcium in. Barnes, C. G., and Greaves, R. I. N. (E.), 130
Thalamus in relation to the cerebral cortex. Walker, A. E. (E.), 229
Thiocyanogen compounds, action of, on nervous system. Moriki, H. (E.), 122
Thyroid gland, role of, in temperature regulation (E.), 583
Time, biological, contrasted with physical (R.), 97
TINGEY, A. H., the iron, copper and manganese content of the human brain, 452
TOD, HENRY, *see* JONES, MAXWELL SHAW.
Tolstoy's " Kreutzer Sonata " and unconscious homosexuality. Velikovsky, P. (E.), 334
Treatment in Psychiatry, by Oscar Diethelm (R.), 572
Tryptophane in cerebro-spinal fluid in tuberculous meningitis. Bock, H. (E.), 133
,, reaction in cerebro-spinal fluid. Spillane, J. (E.), 597
Tuberculoma in corpus callosum and basal ganglia. Trabattoni, C. (E.), 338
Twins, identical, manic-depressive symptoms in (E.), 336
,, investigations on, in epilepsy, 99

Unconscious, the, and reality (E.), 479
Urea clearance test in psychotics, the, by Andrew M. Wyllie, 208
Urethane, effect of, on sigmoid area of cortex and experimental urethane catatonia. Mondio, E. (E.), 103
Urinary retention, neurogenic, effect of pilocarpine on (E.), 483

Vascular disturbances in the brain, the histopathological criteria of, by A. Meyer (Abstract), 509
Vascularity of different areas of the mammalian brain, the relative, by A. C. P. Campbell (Abstract), 510
Vagotropin, Viale's (E.), 128
Vasodilator nerves, action of, and acetylcholine (E.), 584
Vasomotor centre, effect of adrenaline, anæmia and carbon dioxide on. Nowak, J. G., and Samaan, A. (E.), 103
,, control of cerebral blood-vessels, 507
,, disturbances in schizophrenia, a note on, by Louis Minski, 437
,, ,, in mental disorders (acrocyanosis), 408
Vegetative nervous system: action of amines on nictitating membrane. Bacq, Z. M. (E.), 601
,, ,, ,, ,, hypnotics on (E.), 338
,, ,, ,, ,, methoxy-2-iodo-5-phenoxyethyldiethylamine on sympathetic inhibitory system. Bovet, D., and Simon, A. (E.), 601
,, ,, ,, ,, physostigmine, etc., on (E.), 240
,, ,, ,, and hypnosis (E.), 107
,, ,, ,, drugs affecting (mecholyl, etc.) (E.), 603
,, ,, ,, effect of altering conditions of, on choline esterase level of blood, by M. S. Jones and H. Tod, 202
,, ,, ,, in narcolepsy (E.), 237
,, ,, ,, influence of, on metabolism. Abelin, I. (E.), 591
,, ,, ,, mecholyl, benzedrine and other drugs affecting (E.), 238
,, ,, ,, new interpretation of Langley's phenomenon (E.), 602
,, ,, ,, pharmacology of; action of barbiturates (E.), 600
,, reflexes, dynamics of, under conditions of low and high temperatures and of ray energy. Liderman, M. J. (E.), 584
Ventriculography, headache after (E.), 481
Vital staining of central nervous system. Giordano, F. (E.), 241
Vitamin A deficiency, and high cereal intake, lesions of nervous system produced by. Mellanby, E. (E.), 127
,, B deficiency, fat metabolism in nerve tissues of pigeons affected by. Takato, Y. (E.), 244
,, ,, ,, lesions of nervous system in. Davison, C., and Stone, L. (E.), 242
,, ,, in relation to alcoholic polyneuritis, 111
,, B_1 and pyruvic acid oxidation in brain (E.), 596
,, ,, deficiency, potassium in brain in (E.), 126
,, ,, in relation to the catarotulin effect (E.), 592

Digitized by Google

Vitamin B_1, lack of, influence of lactic acid on cerebral respiration of, during. Galvao, P. E., and Pereira, J. (E.), 343
,, C: ascorbic acid content of nervous system. Melka, J. (E.), 126
,, ,, ,, ,, ,, of brain of *Glis glis* during hibernation. Fomin, S. V. (E.), 342
,, ,, ,, ,, in heart. Copello, F. (E.), 126
,, ,, as indophenol-reducing substance in brain (E.), 596
,, ,, content of cerebro-spinal fluid in neurological patients. Heinrich, A. (E.), 344
,, ,, ,, ,, ,, in newborn infants. Rohmer, P., Bezssonoff, N., and Stoerr, E. (E.), 125
,, ,, ,, human brain, demonstration of, by means of dental test. Kalnins, Viktors (E.), 485
,, ,, in cerebro-spinal fluid in normal and pathological conditions. Marinesco, G., Buttu, G., and Olteanu (E.), 126
,, deficiency and nervous system: carotene and vitamin A. Zimmerman, H. M., and Cowgill, G. R. (E.), 127
,, ,, prevention of nutritional encephalomalacia by vegetable oils. Goetsch, M., and Pappenheimer, A. M. (E.), 127
Vitamins, mitochondria and Golgi apparatus. Bourne, G. (E.), 130
Voluntary patient, suicide of, undecided compensation claim arising from, 461
,, patients, observations on, 491
,, ,, *see also under* Mental Treatment Act
WALTER, W. GREY, *see* GOLLA, F. L.
Water metabolism in relation to convulsions. Stone, T. T., and Chor, H. (E.), 596
Weather, The Patient and the, by William F. Petersen (R.), 227
WEICHBRODT, R., On Suicide (R.), 730
WILSON, ISABEL G. H., A Study of Hypoglycæmic Shock Treatment in Schizophrenia (R.) 218
WOOTTON, L. H., and MINSKI, LOUIS, "temporary" treatment: analysis of 30 cases, 366
WYLLIE, A. M., a fatal case of *status epilepticus*, with post-mortem findings, 568
,, the urea clearance test in psychotics, 208

X-ray treatment of cerebral tumours (E.), 482

Young, H. I. P., aspects of temperament in adolescent male offenders, 269

PART II.—AUTHORS REFERRED TO IN THE EPITOME.

Abbatecola, B., 600
Abe, K., 103
Abelin, I., 591
Abuladze, K. C., 104
Acree, T., 114
Adler, E., 592
Aird, R. B., 590
Alagia, D. P., 341
Alexander, F., 334, 479
Alm, O. W., 343
Altenburger, H., 597
Ardy, C., 126
Arkhangelskii, V. M., 342
Arkina, R. Ch., 135
de Arric, 597
Artur, 245
Asarkh, M., 108
Asratzan, E., 103
Axmacher, F., 605

Bacq, Z. M., 601
Baker, A. B., 243
Baldi, F., 125
Barnacle, C. H., 124, 483
Barnes, C. G., 130
Barrera, S. E., 115
Bartolini, B., 128
Baruk, M. H., 481
Baskin, N. A., 110
Battro, A., 239
Bender, L., 606
Benetato, G., 132
Bennett, A. H., 125
Benvenuti, M., 127
Berg, B. N., 591
Bergami, G., 231, 333
Bergonzi, M., 128
Bernard, A., 583
Bezsonoff, N., 125
Birman, B. N., 603
Blackberg, S. N., 121
Blankart, A., 339
Blatt, M. L., 125
Bock, H., 134
Bogolepov, N. K., 107
Bogorad, F. A., 110
du Bois, Cora, 586
Boldyreva, N. V., 592
Bolton, R. O., 237
Bonsignore, A., 126
Boucher, 245
Bouckaert, J. J., 231
Bourne, G., 130
Bovet, D., 601
Bradley, C., 241, 593
Braier, B., 134
Bram, I., 589
Brenner, C., 340
Brewer, E. D., 136
Brewer, E. P., 481
Brickner, R. M., 117
Briscoe, G., 123
Brock, S., 116
Broder, S. B., 342
Brown, G. L., 245
Bruch, H., 606
Brücke, F. T. v., 593
Brundage, J. T., 599
Brusilovskaya, A. I., 120
Budanova, A. M., 596
Budinis, I., 240
Bülow, M., 126

Digitized by Google

Butt, H. R., 132
Buttu, G., 126

Cachera, R., 122
Cahane, M., 345
Cannon, W. B., 582
Carlisle, C. L., 341
Carratala, R. E., 120
Cedrangolo, F., 129, 134
Chang, T. H., 230
Chauchard, P., 602
Chen, K. K., 120
Chevillot, M., 584
Chibnall, A. C., 486
Chor, H., 596
Christoffel, H., 106
Chweitzer, A., 602
Claude, H., 587
Cleveland, D., 582
Cobb, S., 603
Cohen, H., 135
Cohen, I., 117
Cohen, M. E., 603
Cohn, J. V., 239
Colbert, C. N., 111
Cooperman, N. R., 132
Copello, F., 126
Cormulesco, I., 134
Cornwall, L. H., 116
Corteggiani, E., 344, 593
Coulonjou, 245
Cowen, D., jun., 113
Cowgill, G. R., 127
Cox, L. B., 591
Cramer, F., 117

Dameshek, W., 238, 603
Datta, N. C., 346
Daumezon, G., 336
Davidoff, E., 484
Davidoff, L. M., 117, 482
Davis, B. L., jun., 244
Davis, L., 582
Davison, C., 116, 242
Dean, J. P., 483
Deane, J. S., 245
De Biasio, B., 600
Deleonardi, S., 340
De Marco, R., 231
De Nito, G., 604
Detrick, L. E., 599
Dickmeiss, P., 235
Dille, J. M., 240
Dillon, E. S., 109
Dimolescu, P., 346
Dobreff, E., 244
Doshay, L. J., 114
Drabkin, D. L., 339
Drewry, H. H.,
Drury, A. N., 129
Dyer, W. W., 109
Dyke, C. G., 116, 482

Ebaugh, F. G., 124, 483
Eckardt, B., 593, 598
Ehrlich, W., 482
Ellis, L. B., 110
Elsberg, C. A., 482
Elwyn, A., 115
Emdina, S., 108
Engle, B. S., 235
Epel'baum, P. E., 343, 593
Epshtein, S., 593
Epstein, P. D., 590
Euler, H. v., 131, 592
Euzière, 601
Ewalt, J. R., 124, 483

Farago, S., 346
Fauvert, R., 122
Feigenbaum, D., 233
Feldberg, W., 245
Feng, T. P., 333
Fenichel, O., 480
Fenz, E., 606
Ferraro, A., 115
Finkelman, I., 237
Flannery, D. L., 341
Fogelberg, A., 606
Fomin, S. V., 133, 342
Fourt, L., 343
Franklin, C. R., 112
Freud, S., 105, 233
Freund, E., 485
Fribourg-Blanc, 131
Fridman, A. P., 135
Fromherz, K., 340
Frommel, E., 129
Fuchs, S. H., 586

Galli, G., 121
Galli, G. M., 242
Galvao, P. E., 343
Gavrilescu, N., 592
Geblewitz, E., 602
Gel'man, R. M., 343
Gerö, G., 236
Gershanovitch, Z. S., 588
Gévaudan, Mlle., 481
Ghantus, M., 244, 345
Giese, L., 597
Gilarovski, V. A., 119
Giordano, F., 241
Giri, K. V., 346
Girndt, O., 605
Goettsch, M., 127
Goodrich, F. J., 600
Gorodisskaya, H., 593
Greaves, R. I. N., 130
Greenschpoon, R. R., 234
Grinberg, M. D., 588
Gros, O., 605
Gruber, C. M., 599, 601

Halberstadt, G., 337
Harbinson, M. R., 235
Hare, C. C., 136

Hausser, I., 130
Hawking, F., 341
Hayashi, A., 602
Haynes, F. W., 110
Hecker, C. H., 341
Hedström, V., 594
Heimann, M., 485
Heinrich, A., 344
Heldt, T. J., 483
Hempel, J., 597
Hendrick, I., 232
Hennelly, J. T., 341
Herschberg, A. D., 129
Herschberg, R. A., 237
Hetherington, A., 584
Heymans, C., 239
Hofmann, H., 605
Hollombe, B. S., 237
Holthaus, B., 485
Hoseason, A. S., 123
Hrubetz, M. C., 121
Hubbard, R. S., 136
Huddleson, J. H., 480
Hughes, J., 480
Huhn, O., 605

Ichikawa, K., 104
Imber, I., 118
Ingham, J., 487
Ionescu, N., 592
Issekutz, B., jun., 583

Jamieson, G. R., 107
Jedlowski, P., 131
Jervis, G. A., 111
Johnson, T. H., 111
Jolliffe, N., 111
Joltz, S. E., 114
Jones, I., 119
Jones, M. S., 342, 345
Jourdan, F., 105, 231
Jowett, M., 341, 595

Kahane, E., 122
Kalnins, Viktors, 485
Kaminskii, S. D., 604
Kasanin, J., 481
Katz, A. M., 237
Kerr, S. E., 345
King, L. S., 242
Kinnersley, H. W., 126
Knoefel, P. K., 123
Kopet, J. C., 600
Koppanyi, T., 240, 241, 338
Kostakow, S., 133
Kulcsar, M., 128
Kusch, E., 482

Lackey, R. W., 122
Lanari, A., 239
Landesman, J. U., 124
Lapicque, L., 593
Lavergne, V. de, 119
Lazarev, N. V., 120

Digitized by Google

Leese, E. C., 606
Leulier, A., 583
Levy, G., 122
Liber, A. F., 598
Liberson, W., 602
Libman, J., 135
Lichtman, S. S., 128
Liderman, M. J., 584
Linegar, C. R., 240
Lion, E. G., 237
Ljunberg, E., 594
Loewe, S., 603
Loman, J., 238, 603
Luck, J. M., 244
Lufkin, N. H., 243
Luomala, K., 106
Lustig, B., 485
Lynn, E. V., 119

McConnell, K. P., 243, 596
McFarland, R. A., 480
McInnes, R. G., 337
McKendree, C. A., 114
McKinney, J., 114
Maclean, Margaret B., 344
McWilliam, W., 482
Magoun, H. W., 478, 584
Maiorov, F. P., 118
Maiorov, F. D., 604
Maloney, A. H., 123
Maltaner, E. 133
Maltaner, F., 133
Mangelschots, H., 598
Mannini, R., 120
Mansfeld, G., 240
Marchand, L., 589
Marco, R. de, 342
Marinesco, G., 126, 337
Marsovszky, P., 105
Martini, F., 337
Masson, C. B., 112
Meier, C. A., 485
Melka, J., 126
Mellanby, E., 127
Menninger, K., 335
Merritt, H. H., 602
Meyer, Alfred, 109
Milam, D. F., 482
Mingazzini, I., 121
Mitchell, R. S., 484
Mitolo, M., 104, 135
Moffat, W. M., 239
Molitch, M., 587
Mollari, M., 338
Molnar, I., 105
Mondio, E., 103
Moniz, E., 118
Moore, M. T., 124
Moracci, E., 131
Moraeus, B., 338
Moritz, D. U., 128
de Morsier, G., 336
Morton, G., 122
Mosinger, M., 582
Mossa, G., 242
Munteanu, N., 132
Murphy, F. D., 237
Murrell, F. C., 123
Myerson, A., 238

Nagy, M., 344
Nakamura, T., 338
Nattanson, M. H., 238
Nekludov, V., 130
Neustaedter, M., 598
Newman, H., 237
Ney, J., 603
Neymann, C. A., 125, 483
Norcross, N. C., 110
Northington, P., 112
Nowak, J. G., 103

Olteanu, 126
Ormea, A., 336
Osborne, S. L., 125
Ozeretzkovski, D. S., 108

Palcari, A., 346
Palladin, A. V., 230, 343
Pappenheim, E., 597
Pappenheimer, A. M., 127
Parhon, C. I., 345
Paulian, D., 124
Pelliecioli, V. G., 241
Peneff, L., 244
Penfield, W., 110
Penrose, L. S., 232
Peola, P., 229
Pereira, J., 343
Perutz, A., 130
Peters, R. A., 133, 596
Petrova, V. V., 135
Pette, H., 345
Piper, P., 239
Piper, S. H., 486
Plaut, F., 126
Polak, J. J., 592
Pool, J. L., 591
Porta, V., 241
Potts, H. D., 111
Prinzmetal, M., 484
Prior, G. P. U., 607
Pugh, C. E. M., 595
Putnam, T. J., 478, 602

Quastel, J. H., 341, 487, 595

Radici, G., 120
Rafkin, I. C., 235
Randall, W. A., 338
Ranson, S. W., 584
Rashba, E. Y., 343
Rashba, H., 230
Rashba, O., 131
Rausnitz, E., 344
Ravdin, I. S., 339
Raven, J. C., 232
Raymond-Hamet, 601
Read, C. F., 607
Reifenstein, E. C., jun., 484
Reitler, R., 597
Retchnik, M. A., 110
Richter, G. E., 604
Riggs, H. E., 109
Riley, H. A., 116
Roboz, P., 344
Rohmer, P., 125
Romesco, P., 134
Rose, C. L., 120
Rosenblueth, A., 582
Rosenthal, F., 120
Roussy, C., 582
Rubinstein, H. S., 230
Russell, N. G., jun., 136
Russell, N. M., 136

Sack, G., 600
Salkan, D. M., 590
Salmon, A., 242
Samaan, A., 103
Saul, L. J.,
Scarff, J., 114
Schaeffer, R. L., 346
Schilder, P., 334
Schlientz, W., 485
Schmitt, F. O., 343
Schram, M., 338
Schube, P. G., 243
Schweitzer, A., 333, 339, 604
Selzer, H., 335, 336
Seo, M., 103
Serban, F., 134
Seuberling, O., 598
Shapiro, L. B., 607
Shen, S. C., 333
Shimada, S., 344
Shirokov, A. A., 589
Silver, G. A., 122
Sinclair, R. G., 596
Simon, A., 601
Sinclair, R. G., 243
Siwe, S., 134
Sjöden, B., 587
Skinner, E. F., 486
Skipetrov, A. I., 107
Sklar, N. I., 588
Skvortzow, K. A., 107
Snachkova, A. A., 132
Sokolanski, G. G., 584
Solomon, P., 484
Sparrow, W. J., 585
Speirs, G. O., 338
Speirs, R. E., 338
Spillane, J., 597
Stedman, E., 593
Stedman, E. and E., 595
Stepanov, P. N., 604
Stern, E. L., 128
Stern, I. B., 110
Stoerr, E., 125
Stone, L., 242
Stone, T. T., 596

Digitized by Google

Stookey, B., 114
Strashesko, D. N., 133
Stratman-Thomas, W. K, 482
Straub, J., 344
Strecker, E. A., 480
Swanson, E. E., 239

Tai, F. I., 230
Takata, M., 134
Takato, V., 244
Takeuchi, S., 123
Tanasesco, G., 124
Tarsis, F. I., 124
Taylor, H. W., 338
Taylor, N. M., 346
Taylor, W. F., 122
Tennenbaum, M., 487
Tiffeneau, M., 240
Tihen, H. N., 599
Tilney, F., 113
Timme, W., 113
Tod, H., 132, 342, 345, 597
Toffelmier, G., 106
Tomescu, P., 345
Tomioka, T., 129
Tornu, A., 241
Tournade, A., 584
Trabattoni, C., 337
Tropp, C.. 593, 598
Turkevitch, O. M., 588
Tyukody, F. v., 240

Uiberrak, K., 606
Ujsaghy, P., 135

Vainberg, I. S., 603
Vialetto, E., 109
Vikker, J. L., 109
Voronova, M., 108

Wada, M., 103
Wadsworth, A., 133
Wahlström, G., 594
Walker, A. E., 229
Wallach, G., 120
Wechsler, I. S., 111
Weil, A., 485
Weil-Matherbe, H., 488, 594
Weiss, S., 236
Werner, A. A., 118
Westenbrink, H. G. K., 592
Wheatley, A. H. M., 487
Whitehead, W. S., 483
Whitnak, C. H., 343
Wichmann, B., 485
Williams, E. F.,
Wispelaere, M. de, 482
Wittkower, E., 244
Wolf, A., 113
Wright, Samson, 333, 339, 604

Yarkin, J. C., 479
Young, F. G., 596
Yuskin, J. C., 585

Zimmerman, H. M., 127
Zinkin, A. M., 583
Ziskind, E., 237
Zolotova, K. A., 583

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CONTENTS FOR NOVEMBER, 1937

Original Articles— PAGE

Convulsion Therapy in Schizophrenia; by *Alexander Kennedy, M.D., M.R.C.P., D.P.M.* 609

An Unusual Complication of Insulin Shock (Hypoglycæmic) Therapy; by *C. B. Molony, M.B., D.P.M., and M. S. Honan, M.A., M.D.* . . . 630

The Value of Continuous Narcosis in the Treatment of Mental Disorder; by *Harold A. Palmer, M.D., M.R.C.P., D.P.M.* 636

Epileptic Reactions: An Attempt at Classification; by *Arthur Guirdham, M.A., D.M., B.Sc.Oxon., D.P.M.Lond.* 679

The Treatment of Epilepsy with Psychosis by Prominal; by *D. E. Sands, M.R.C.P.Ed., D.P.M.Lond.* 703

Familial Psychosis Associated with Endocrine Disorder; by *T. A. Munro, M.B., Dipl. Psych., M.R.C.P.Ed.* 707

Clinical Notes and Cases—

A Case of Profound Dissociation of the Personality; by *C. L. Copeland, M.B., Ch.B., D.P.M., and E. Howard Kitching, M.D., M.R.C.S., D.P.M.* 719

Reviews 727

Convulsion Therapy in Schizophrenia [Die Konvulsionstherapie der Schizophrenie]; by *Ladislaus von Meduna.*—The Growing Child and Its Problems; by *Emanuel Miller.*—Reading, Writing and Speech Problems in Children; by *Samuel T. Orton.*—On Suicide [Der Selbstmord]; by *R. Weichbrodt.*

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